

**A TEXT-BOOK
OF X-RAY DIAGNOSIS
BY BRITISH AUTHORS**

‘What shadows we are, and what shadows we pursue.’

EDMUND BURKE, Sept. 9th, 1780.

A TEXT-BOOK OF X-RAY DIAGNOSIS

BY BRITISH AUTHORS

IN THREE VOLUMES

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PUBLISHERS' NOTE

THE size of the volumes in this reprint has been reduced in order to comply with the regulations now in force prescribing the area of the type surface in relation to the size of the page.

The type area is the same as in the first issue and the reduction in size has taken place at the expense of the margin.

The Publishers regret this reduction, one which somewhat mars the appearance of the volumes, and they hope that it will be accepted as a war-time necessity. When a new edition of the work is called for it will be their endeavour to return to the original format.

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P R E F A C E

THE OBJECT of the Editors in presenting this text-book is to provide within reasonable limits a comprehensive survey of the present position of X-ray diagnosis. Diagnostic radiology is becoming an increasingly complex specialty, and it is difficult for one person to be equally expert in all its branches. The editors are fortunate therefore in having the help of collaborators, both radiological and clinical, who are distinguished in particular branches of the subject. It is hoped that this has made the work the more authoritative, and that it will be of value not only to the post-graduate student of radiology, but also to the clinician. In conformity with this design only essential details of technique are included, and the subject of X-ray physics is not dealt with.

For convenience of reference, the work is published in three volumes, each containing as far as possible subjects of allied interest. Thus Vol. I deals mainly with the thorax, Vol. II with the abdomen, and Vol. III with the skeletal and nervous systems.

It is not possible, even within the generous limits allowed by the publishers, to illustrate every condition demonstrable by radiology, but the illustrations chosen are, it is hoped, representative, and give due emphasis to the common lesions met with in radiological practice. Considerable interchange of material for illustrations has taken place between the various contributors to the book, and the Editors are greatly indebted for the loan of illustrations from other colleagues, detailed acknowledgments of which will be found at the beginning of each volume. They are also grateful to Mr. Boutall, of Messrs. Vaus & Crampton, for the care and attention he has given to the preparation of the blocks, and to Messrs. Hazell, Watson & Viney for their careful work with their printing.

Finally, the Editors desire to express their sincere thanks to the publishers, and in particular Mr. H. L. Jackson and Mr. F. Boothby, for their co-operation and advice, without which this book could not have come into being.

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VOLUME I

PART ONE

CARDIO-VASCULAR SYSTEM

BY

PETER KERLEY, M.D., F.R.C.P., F.E.R., D.M.R.E.

A TEXT-BOOK OF X-RAY DIAGNOSIS

PART ONE

CARDIO-VASCULAR SYSTEM

CHAPTER I

X-RAY EXAMINATION OF THE HEART

X-RAY EXAMINATION of the heart and great vessels has not materially altered clinical and pathological methods of investigation. It is impossible by radiology of the cardio-vascular system to arrive at a decisive diagnosis comparable, say, with the X-ray diagnosis of gastric ulcer or phthisis. Cardiac radiology is not and never can be a short cut to diagnosis, and X-ray findings are of value only when correlated with clinical findings.

The intrinsic movements of the heart and the alterations produced on it by adjacent moving organs, the lungs and the diaphragm, are a serious obstacle to detailed investigation of its function by X-rays. Cardiac cinematography and Röntgen kymography, although partly overcoming this obstacle, have not yet contributed materially to the investigation of heart disease. The methods of X-ray examination in common use are fluoroscopy, orthodiagraphy, and teleradiography.

FLUOROSCOPY

Screen examination is of the greatest importance. It gives to the observer an immediate knowledge of the relationship of the heart to the neighbouring intrathoracic organs. He can see at a glance whether the shape or position of the heart is altered by such extrinsic factors as scoliosis or fibrosis of the lungs. He has a clear view of the apex of the heart through the gas bubble in the stomach, and if necessary he artificially increases the size of the gas bubble by giving the patient an effervescent mixture to drink. Under the screen the patient can be slowly rotated into the most suitable positions for examining the various chambers, and in many cases valuable information can be obtained by watching the passage of a barium bolus through the oesophagus. The disadvantages of fluoroscopy are that there is no permanent record of the appearances and that fine changes in the pulmonary vessels cannot be detected.

ORTHODIAGRAPHY

This is a method of estimating the radiological size of the heart. The central ray, i.e. that originating from the centre of the target, does not diverge. It follows that if this ray alone can be employed, fallacies due to divergence of the radiation can be eliminated. An instrument known as an orthodiagraph has been devised for this purpose. In the orthodiagraph the X-ray tube is *entirely independent of the screen, and the tube can be moved freely upwards, downwards, and laterally.* A fine diaphragm is fitted, and this is contracted so that only the central ray gets through. The patient is placed behind the screen, which is pressed tightly against his chest, and when the tube is activated the central ray appears as a tiny beam of fluorescence through the air-containing lung. This tiny beam is carefully moved around the cardiac silhouette, and at selected points the outline is marked on the screen with a grease pencil. The largest possible silhouette is drawn, i.e. ventricular diastole for the heart area, and ventricular systole for the vascular area. During the examination the patient breathes as quietly as possible. The drawing of the diaphragm is made so far as possible during the time of respiratory pause. The final drawing is traced from the screen on to transparent paper which serves as a permanent record. Measurements of the heart shadow made with an orthodiagraph are more accurate than those made on a teleradiograph. On the other hand, in any fluoroscopic examination the personal factor is of great importance, and in the same subject two independent observers may record widely different measurements.

TELERRADIOGRAPHY

The greater the distance between the source of the radiation and the object radiographed, the less the distortion due to divergence of the rays. At 2 metres distance the radiographic size of the heart is about 1 cm. greater in width than the orthodiagrammatic measurement. At 3.5 metres distance the measurements on the film are equal to those made with an orthodiagraph. The teleradiogram has, of course, the great advantage that it provides a better record of the heart in relation to neighbouring organs than the orthodiagram. It also allows of detailed study of the pulmonary vessels, and the personal equation does not enter to the same degree that it does with orthodiagraphy. *Its disadvantages are (a) expense and the large amount of space taken up by high-powered apparatus, and (b) the difficulty of reproducing all factors exactly at different intervals. These latter disadvantages are being overcome by modern methods of construction.*

close the tube current by the pulse at the wrist or by the heart sounds. Using these devices, it is possible to determine in which phase of the cardiac cycle the radiogram has been made, but for all practical purposes a radiogram can be made without these devices if the exposure is made at the end of expiration. To ensure good detail in the oblique and lateral views it is often necessary to take pictures in full inspiration, but so far as possible this should be avoided. No fixed angle can be given for the oblique views, and in every case the patient must be screened into the correct position.

CINERADIOGRAPHY

This branch of radiography has made remarkable progress in recent years. In this country *Russell Reynolds* has perfected a simple apparatus whereby the screen image is photographed. There is no doubt but that cineradiography has a great future in cardiology, but at the present time it suffers from two disadvantages: (a) the time factor is too slow, i.e. the number of pictures taken per second is too small and bears no relation to the cardiac frequency; (b) the pictures are taken at a short distance.

KYMOGRAPHY

This method has come to the fore recently and gives a graphic registration of a continuous movement. The method was first suggested by *Sabat* of Moscow, was later improved by *Knox* of London, and has recently been perfected by *Stumpf* and *Cignolini*. The X-ray kymograph consists of a metal grid with a row of transverse slits of equal width and equidistant from each other. The grid is placed between the patient and the film cassette, and during the exposure either the grid or the cassette is moved at right angles to the slits in the grid. Using the moving-grid multiple-slit method, the kymographic picture of the heart is similar to that of the still-heart shadow, but the outlines of the shadow have a serrated outline instead of the normal smooth contour. This type of kymogram is read from above down. Using the fixed-grid and the moving-cassette method, the outline of the shadow is broken, and each section represents the movements of one point in the beating heart. This type of kymogram is read from below upwards and gives an accurate picture of time relationship between the various sections of the heart. The kymograph has proved useful in demonstrating pericardial adhesions at the base and in differentiating between acquired and transmitted pulsation. The kymograph has not so far been very extensively used in cardiology and so its full value cannot be estimated.

CHAPTER II

THE NORMAL CARDIO-VASCULAR SHADOW

NORMAL CONTOURS

In the Postero-anterior View the normal cardio-vascular shadow is of three types—the long or narrow, the oblique or globular, and the transverse. The type of shadow is largely dependent on the shape of the chest—in a long narrow chest the shadow is usually of the narrow type, and in a short broad chest the shadow is usually of the globular or transverse type. In young infants the heart is always of the transverse type, and gradually alters in shape over a period of years; first tending to become narrow, later widening a little, and not definitely reaching its adult type until after puberty.

THE RIGHT BORDER OF THE CARDIO-VASCULAR SHADOW consists of two distinct shadows separated by a clear-cut notch. The lower and denser of these shadows is the outline of the right auricle. Recent work with the X-ray kymograph shows ventricular serrations on the lower border of this shadow, and it has been suggested that the right ventricle does project out into the right cardio-phrenic angle. This view is not generally accepted, and it is assumed that these ventricular serrations are transmitted from the right ventricle.

The upper shadow on the right side consists in its lower part of the superior vena cava and the ascending aorta. The aorta rapidly turns into the central shadow while the border of the superior vena cava can be clearly followed almost as far as the clavicle. The innominate vein probably plays some part in the formation of this upper shadow. In the normal adult chest the ascending aorta does not project outwards over the shadow of the superior vena cava.

THE LEFT CARDIO-VASCULAR SHADOW consists of three and sometimes four distinct parts. The lowest and largest of these is formed by the left ventricle. Above this a slight projection is sometimes seen, more often on the screen than on the film. This projection is formed by the appendage of the left auricle. The shadow now recedes inwards to form a flat rather convex area formed by the conus pulmonalis and the pulmonary artery. Finally we have the dense aortic knuckle formed by the aortic arch seen obliquely.

THE NORMAL DESCENDING THORACIC AORTA is of two types. In one its whole course is in front of the vertebræ, and it is therefore invisible. In the second type it runs slightly to the left of the spine and can be seen as a straight line running downwards, parallel to the spine. The normal aorta increases slightly in size and position with age. In elderly people the whole of the thoracic aorta tends to swing round to the left and unfold. Thus we find in

old age the ascending part of the aorta jutting out on the right side, the aortic knob higher and more to the left, and the descending aorta seen as a convex shadow well to the left of the middle line and clearly visible through the normal heart shadow.

The right main branch of the pulmonary artery projects outwards just above the notch dividing the two main shadows of the right auricle and the superior vena cava. The left main branch of the pulmonary artery is shorter and always higher than the right. The pericardium is normally never visible.



FIG. 1.—Normal heart of a boy aged 14. Radiogram at 2 metres. Note the position of the normal apex through the gas-distended stomach.

In the left cardio-phrenic angle there is a small triangular opacity formed by a pad of fatty tissue at the pleuro-pericardial reflection. In the right cardio-phrenic angle the inferior vena cava is often seen as a fine triangular opacity, but on this side also some fatty tissue may be present and cast an appreciable shadow.

In all radiological examinations of the heart oblique views are essential. These oblique views often give more decisive evidence than the postero-anterior view, hence they should be taken with great care. The popular idea that oblique views can be taken by rotating the patient 45 degrees to either

side is fallacious. The degree of rotation required varies with each case and can be estimated only by first screening the patient into the correct position.

The **First or Right Oblique View** is obtained by rotating the patient to the left so that his right shoulder is touching the screen. If the vascular shadow is the part required in most detail, the patient is turned until the pulmonary artery is seen end-on and the inferior contour of the whole aortic arch becomes visible. If the left auricle is to be studied, the patient is rotated until there is a translucent space visible between the posterior part of the heart shadow and the spine. This clear space is often referred to as Holzknicht's space. It is encroached on and often obliterated by enlargement of the left auricle or general enlargement of the heart.

In this first oblique view the heart is seen as a pear-shaped opacity (see Fig. 2). *The anterior contours* from below upwards represent the following structures: 1, the left ventricle; 2, the conus pulmonalis; 3, the division of the pulmonary artery; 4, the anterior border of the ascending aorta; 5, the left subclavian artery, the shadow of which can be seen rising over the apex of the lung. *The posterior contours* from below upwards are as follows: 1, the inferior vena cava; 2, the edge of the left auricle; 3, behind 1 and 2, the anterior edge of the descending thoracic aorta; 4, the left main bronchus; 5, the descending part of the arch; and 6, the superior vena cava. The trachea is clearly visible running downwards over the shadow of the aortic arch.

The **Second or Left Oblique View** is taken by rotating the patient to the right side so that the left shoulder is touching the screen. Again the degree of rotation varies according to whether the aorta or the ventricles are to be studied (see Fig. 3).

In this position *the anterior contours* from below upwards are as follows: 1, the edge of the right ventricle; 2, the appendage of the right auricle; 3, the ascending part of the aorta; 4, the superior vena cava and the innominate vein. Posterior to 4 is the trachea. *The posterior contours* from below upwards are: 1, the left ventricle; and 2, a small part of the left auricle. Posterior to 1 and 2 the anterior part of the descending thoracic aorta is visible. Above 1 and 2 there is a wide translucent space whose upper border is formed by the inferior margin of the aortic arch. This space is known as the aortic window, and the left main branch of the pulmonary artery can be seen running across the middle of it. Above the aortic arch there is another small translucent space known as the aortic triangle. This triangle has as its base the top of the aortic arch; its anterior side is formed by the shadow of the left subclavian artery, and its posterior side by the spine. The apex of the triangle is at the level of or just above the clavicle, where the left subclavian artery is running across the apex of the lung and in this view appears to cut across the spine.

The **Position of the Œsophagus** relative to the cardio-vascular structures should be studied carefully. The normal Œsophagus in its course through the

chest is in close contact with the aortic arch, the left main bronchus, the left auricle, and the descending thoracic aorta. These structures produce visible impressions on the oesophagus. The radiological anatomy has been elaborated in great detail by *Evans*, who has recently published an excellent monograph

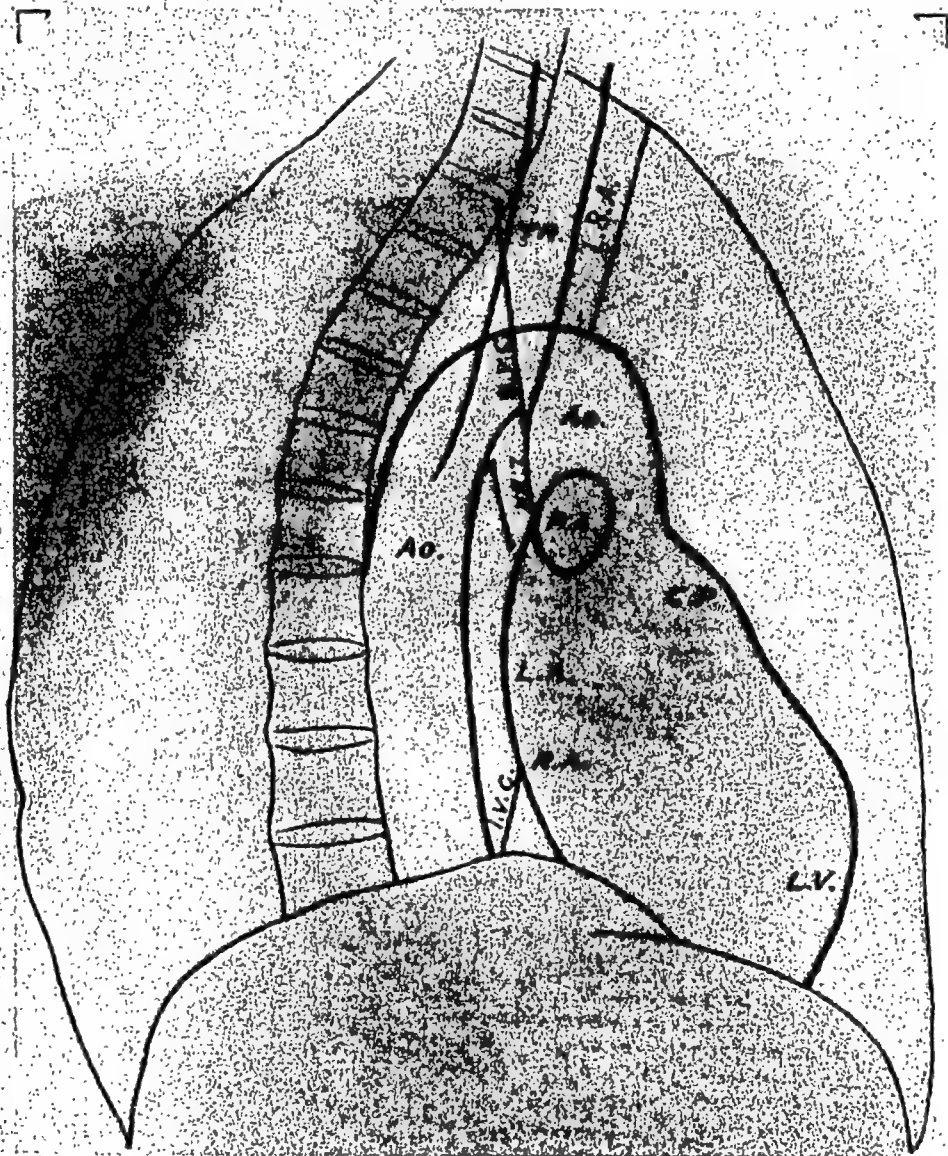


Fig. 2.—The first oblique view.
 L.V. Left ventricle. C.P. Cónus pulmonalis. Ao. Aorta. P.A. Pulmonary artery. TR. Trachea. S.V.C. Superior vena cava. L.B.R. Left main bronchus. R.A. Right auricle. I.V.C. Inferior vena cava. L.S.A. Left subclavian artery.
 on the subject. From left auricle down R.A. Right auricle. phag. follows: (1) The aortic arch produces the deepest impression on the phagus. This impression is best seen in the postero-anterior and lateral views, and in the former view use is made of it to measure the

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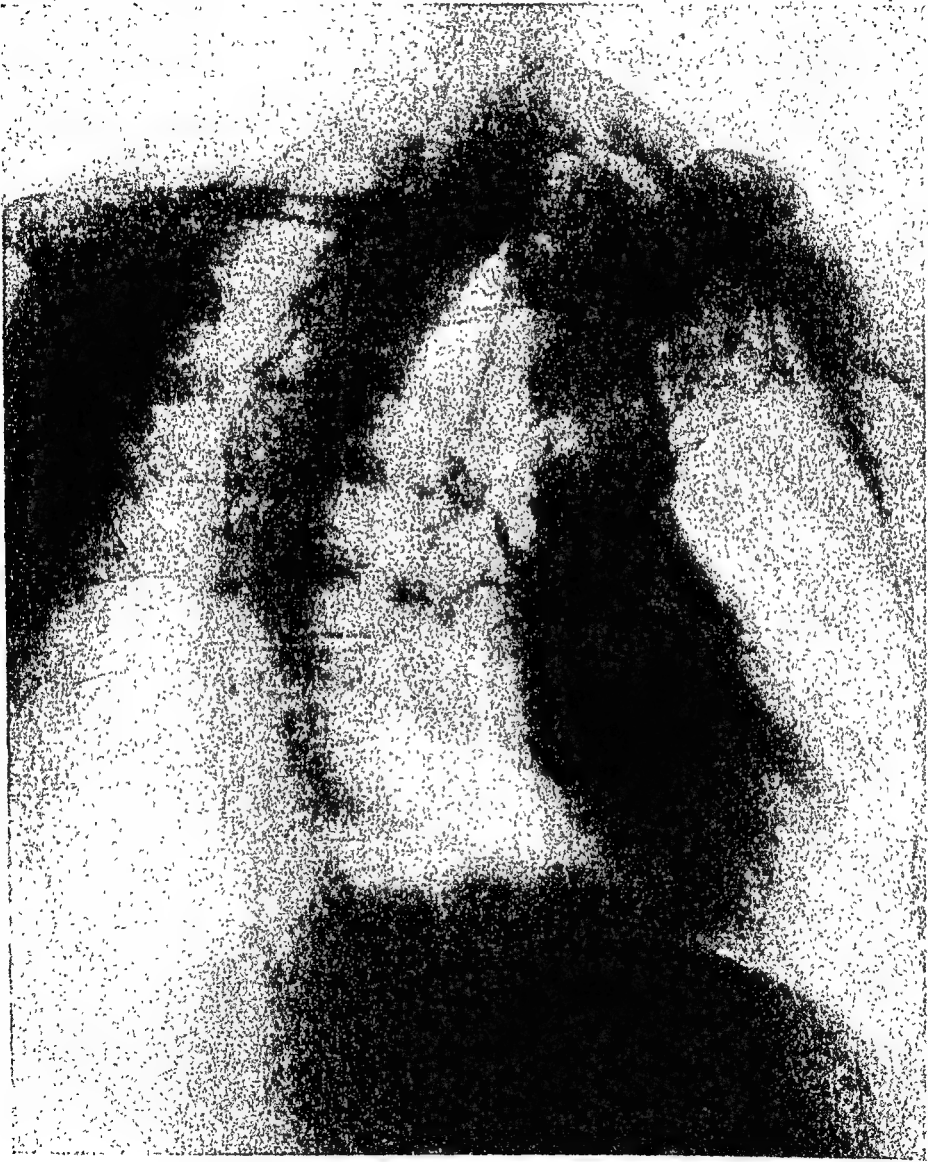


FIG. 2.—The first oblique view.

on the subject. From above downwards the œsophageal impressions are as follows: (1) The aortic arch produces the deepest impressions on the phagus. This impression is best seen in the postero-anterior and first oblique views, and in the former view use is made of it to measure the width of

aorta. (2) The left main bronchus produces a slight impression on the œsophagus. (3) The normal left auricle, especially in young children, produces an obvious impression. This impression is greater in expiration than in inspiration. In individuals with a long narrow chest and heart, this impression



FIG. 3 — The second oblique view

may be completely absent. (4) Just above the diaphragm the œsophagus crosses in front of the descending aorta, which makes a slight indentation on the œsophagus. In the postero-anterior and first oblique views these normal impressions are all concave to the left.

Just below the left diaphragmatic shadow—draw a line to join the notch separating the right auricular and ventricles. This line is known as LD, the *long or oblique diameter* of the heart. The average length of LD in the adult male is 13.6 cm., and it varies normally between 11 and 15.5 cm.

The *transverse diameter* of the heart is obtained by selecting the two widest points on each border and joining them at right angles to the central perpendicular line. The sum of

these two distances is the transverse diameter. It varies normally between 9.2 and 14.5 cm. in the adult male, and the average measurement is 12.2 cm.

The *broad diameter* of the heart BD is obtained by joining the lowermost point of the right cardio-phrenic angle and the uppermost point of the left auricular shadow at right angles to the central perpendicular line.

The sum of these two distances is the broad diameter. In the normal adult male the average measurement of BD is 18 cm., and it varies between 16 and 20 cm.

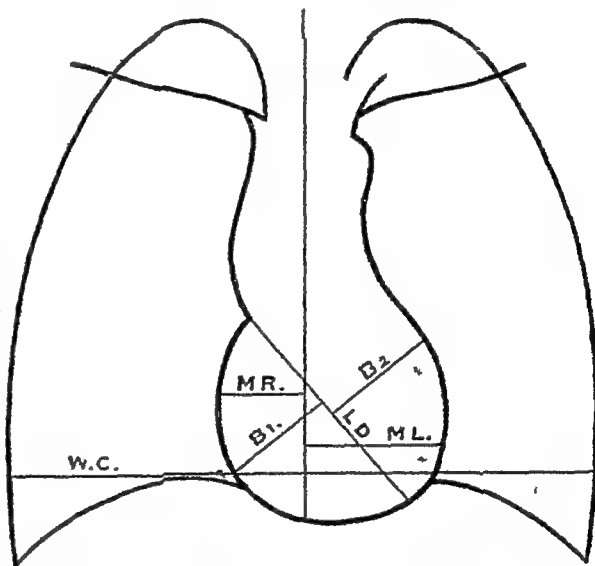


FIG. 4—Cardiac measurements in the postero-anterior view.

The *heart lung coefficient* is the ratio between TD, the transverse diameter of the heart, and a line drawn across the widest diameter of the chest. This ratio is as 1 is to 2 in the normal adult male.

The *antero-posterior diameter* or horizontal depth of the heart is measured in the true lateral position. It is obtained by drawing a line from the point where the anterior borders of the heart and sternum meet to the point of maximum convexity on the posterior surface of the heart. The average measurements of this diameter for the adult male are 9.4 cm., and for the adult female 8.3 cm.

The *lateral diagonal diameter* is more difficult to estimate. This mea-

ment is also made in the true lateral view, and constitutes the distance between a point taken from the apex of the angle formed by the sternum and the diaphragm and a point taken between the two shadows of the pulmonary artery and the pulmonary veins.

Various workers have attempted to estimate the volume of the heart by multiplying the following three diameters TD and LD in the postero-anterior

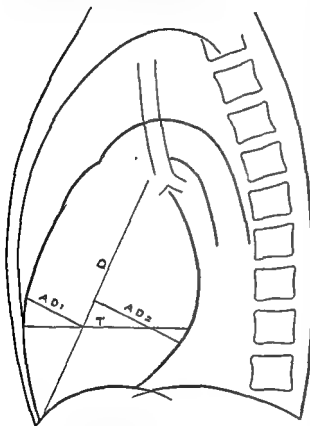


FIG. 3—Cardiac measurements in the lateral view

view, and T in the lateral view. Roesler has done some remarkably detailed work on volumetric analysis this should be studied in the original. Roesler's suggestion that the area of the cardiac silhouette is the size of the closed fist is surprisingly accurate and most useful for practical purposes.

The width of the aorta is measured by filling the oesophagus with barium and estimating the distance between the widest point on the aortic knob and the point of greatest deviation of the oesophagus by the normal aorta.

Two mm. is deducted from this figure to allow for the thickness of the oesophageal wall between the barium and the aorta. In the normal adult male the width of the aorta so measured is 2.5 cm., and it varies

between 2 and 2.5 cm. In the case of an unfolded aorta this measurement will obviously be greater, but this difficulty can be overcome by rotating the patient slightly until the aortic axis is back to its pre-unfolded position.

It must be emphasised that measurements of the radiological heart shadow are in no sense measurements of the real size of the heart. The measurements have proved of most value in estimating alterations in size of the heart shadow due to physiological or toxic causes.

THE NORMAL CARDIAC PULSATION

The pulsation of the heart as seen on the screen is of considerable value, and may give decisive evidence in diagnosis. Normally the pulsations on the *left border* are much more vigorous than on the right. The most obvious pulsation is noticed on the left border of the left ventricle. During systole the left border is seen to contract forcibly, and during diastole it moves outwards. The range of excursion in the average normal varies from 2 to 4 mm. Immediately following the left ventricular contraction the shadows of the pulmonary artery and the aortic knob bulge forcibly outwards. If the eye is, so to speak, centred between the left ventricular border and the aortic knuckle, a 'see-saw' movement is observed, the knuckle coming out as the ventricle goes in.

It is usually impossible to see any pulsation over the small area of the left border formed by the left auricular appendage; if, however, the auricular-ventricular interval is prolonged, a slight contraction of the appendage may be seen preceding the contraction of the left ventricle.

On the right side the lower border formed by the right auricle shows a faint auricular contraction, the range of excursion being no more than 1 mm. In cases of hypertrophy of the right ventricle, however, forcible ventricular pulsation may be transmitted to this right border. Although the true edge of the right vascular shadow is formed by the superior vena cava, it usually shows in its lower part a vigorous aortic pulsation which is transmitted to the superior vena cava by the ascending part of the aorta.

Pulsation is greater in children than in adults, and is increased by exercise.

PHYSIOLOGICAL FACTORS INFLUENCING THE SHAPE AND SIZE OF THE HEART

The size of the normal heart is dependent on the weight of the body, the age of the subject, and the amount of blood. The radiological size and shape are dependent on many other factors most of which have no influence on the function of the heart. These factors are the shape of the bony thorax, the position of the diaphragm, and the slackness or rigidity of the mediastinum as a whole.

The Infant's Heart

The heart of an infant is of the transverse type and appears very large relative to the transverse diameter of the chest. This increase in size, partly real and partly apparent, is due to the high position of the diaphragm, which is raised by the normally large infantile liver and the normally gas-distended infantile stomach. In children up to the age of 6 or 7 years the heart is capable of very wide variations in size. It has frequently been observed that children at the end of a prolonged expiration, such as occurs during crying or at the end of a whoop, almost completely empty the lungs of air. Radiograms show both lungs almost completely opaque and the heart shadow enlarged to both sides, and, on the left side, almost touching the axillary line. Subsequent radiograms

taken in inspiration show a normal heart shadow. This is a point of considerable importance, as not infrequently these normal deviations in size lead to erroneous diagnoses of cardiac enlargement. It is difficult to make accurate



FIG 6—Teleradiogram of a normal infant's chest, taken at the end of expiration. Note the considerable deflation of the lungs and the apparent enlargement of the heart.

orthodiagrammatic measurements in young children, and in the author's experience teleradiograms are more reliable.

The Child's Heart

The heart may show considerable variations in shape until the age of puberty has been passed. It is no uncommon thing to see a transverse heart at the age of 5 become a narrow or oblique heart by the age of 10. This is due to the frequency of respiratory infections in young children. Such infections, if severe, are usually accompanied by compensatory emphysema, which persists for some time and leads to permanent alteration in the shape of the chest.

The Adult Heart

After the age of puberty the normal radiological heart shadow falls into one of three groups: the narrow heart, the oblique heart, and the transverse

heart. The most important fact in determining the shape appears to be the width of the chest. Thus in individuals with a long and narrow chest, we see a small narrow heart, and in individuals with a wide chest we see the transverse type of heart. The average individual has an oblique heart. In the average case the ratio of the transverse diameter of the heart to the maximum width of the chest is about 1 to 1.9 or 1 to 2. Since the heart shape is dependent to a large extent on the shape of the chest, this coefficient is also a fairly reliable guide to size.

During full inspiration the heart decreases in size, and during full expiration it increases in size. This effect is enhanced by the fact that the heart rotates as the diaphragm descends and ascends. It follows that conditions which raise or depress the diaphragm will cause an alteration in the shape of the heart, and the presence or absence of such factors should always be determined. Pregnancy is the only important physiological factor causing marked elevation of the diaphragm, and all women in the later stages of pregnancy show a transverse type of heart. During pregnancy the normal lumbar lordosis is increased and the shape of the chest consequently alters. This postural change also tends to make the heart appear more transverse. The common pathological causes of altered position of the diaphragm are ascites and emphysema.

There has been considerable discussion as to whether a *small radiological heart shadow* is pathological or not. *Wenckebach* published one unique case in which the heart was apparently hanging free in the chest and had no diaphragmatic attachments. This was undoubtedly a congenital malformation. Exhaustive autopsy investigations have shown that the small radiological heart shadow is not pathological. Even during the course of wasting diseases there is usually no X-ray evidence to show that the heart is becoming smaller, although autopsy may show an atrophic heart.

It must not be inferred because a heart appears small that it cannot be the subject of acquired or congenital disease. Both in mitral stenosis and many congenital lesions the heart shadow may be of the small type, but in these cases there is usually other evidence, such as disproportion in the size of different chambers, which reveals the pathology.

In the *average or oblique type of heart*, two-thirds of the shadow lie on the left side and one-third on the right side of the spine. A line drawn from the aortic knuckle to the edge of the left ventricle usually shows inside this line at the level of the pulmonary artery an inch or two of translucent lung tissue. As will be shown later, this line does not enclose lung tissue at any point in certain diseases of the heart, notably mitral stenosis and hypertension with congestive failure. There is a tendency to describe this finding as "mitralisation of the heart," and generally speaking the appearance signifies some pathology in the heart. In a small percentage of normal oblique hearts, the pulmonary artery shadow is rather prominent and results in this so-called mitralisation appearance. A similar

appearance can be produced by scoliosis or fibrosis of the left lung, and it cannot be too strongly emphasised that "mitralisation" by itself is no evidence of disease of the heart. There must be additional evidence either clinical or radiological to justify this appearance as X-ray evidence of disease.

The transverse type of heart is usually seen in obese short-necked individuals. The diaphragm in these individuals is usually at the level of the 5th interspace anteriorly, and may be as high as the 4th. The heart appears squat and broad, its outlines are not very sharply defined, owing to the secondary radiation from the excessive fat on the chest wall, and its pulsation appears relatively feeble. In the normal obese individual the ratio between the transverse diameter of the heart and the maximum width of the chest wall remains within the normal limits of 1 to 1.9, but errors may be made if there is an excessive deposition of fat in the pleuro-pericardial reflection. This deposition of fat may closely simulate the apex of the heart, and there may be well-marked transmitted pulsation visible on it. Careful screen examination with a small diaphragm is the best method of estimating the true source of the apex, but fairly hard teloradiograms or radiograms taken with a Lysholm grid are also effective in showing up the true cardiac outline through this fat. The aortic shadow in obese individuals also appears abnormally wide because, while the aorta is normal in length, it is compressed into a smaller space. Dilatation of the aorta should never be diagnosed on a postero-anterior view alone, and in obese individuals the normal aortic outline should be identified in the oblique views.

The Effect of Excessive Exercise on the size of the heart has long been a subject of contention. The majority of workers now believe that excessive muscular exercise cannot by itself produce a permanent pathological enlargement. It is even doubtful if temporary enlargement occurs during exercise, as it is impossible to estimate the size of the heart during exercise. In most athletes the heart shadow is rather larger than the normal average, but careful measurements usually show the relative proportions to be within the normal limits, and the apparently large size is simply one aspect of a general muscular development. In doubtful cases the only safe procedure is for one observer to make repeated measurements over a period of three months, and if all the measurements are the same, the heart can be said to be normal.

The Effect of Drugs on the heart has been studied by many authors. Amyl nitrite and atropin are said to cause a slight decrease in size. Digitalis and salyrgan may produce a minimal decrease in size in cases of congestive heart failure, but many authors, including *Parkinson*, are sceptical of this.

Generally speaking, most workers are sceptical as to the possibility of temporary wide alterations in the size of the heart either from physiological or pathological causes. Isolated reports of such temporary enlargement are not infrequent, but the possibility must always be borne in mind that these reports deal with cases in which the heart was not normal to begin with.

CHAPTER III

DISPLACEMENT OF THE HEART

THE MOBILITY OF THE HEART AND MEDIASTINUM

THE NORMAL heart and great vessels are not immobile structures in the chest. The heart is capable of direct lateral movement to either side, and can also rotate to either side. The aorta as a whole shifts to the left during normal pulsation, and our knowledge of intrathoracic tumours shows that it can be displaced upwards, downwards, and to either side. The aorta should not be thought of as a rigid tube in the chest, but rather as a tense coiled spring, capable of unfolding, stretching, and kinking when its elasticity is interfered with. The degree of mobility of the heart and mediastinum varies enormously in different individuals, and we have no means of estimating in the normal individual how slack or how rigid these structures are, except by inducing a pneumothorax. The movement of the mediastinum has been studied in great detail in tuberculous patients treated by pneumothorax and phrenic avulsion, and we find that about 50 per cent. of normal individuals have a slack mediastinum and 50 per cent. a rigid mediastinum. In those with a slack mediastinum the heart can be pushed to one or other side as much as 3 to 4 cm. and can rotate to the left about 30 degrees. Its possible rotation to the right is less, as such rotation is restricted by the superior and inferior venæ cavæ and the ascending part of the aorta. In these individuals the degree of displacement or rotation can be estimated by observing the position of the trachea, which is more loosely attached than the vascular structures and swings freely to either side. In individuals with a rigid mediastinum all the mediastinal structures are practically immobile, and in cases of excessive pressure on one or other side pseudo-herniation of the lung may take place at the level of the 3rd or 4th dorsal vertebra, where there is normally some loose areolar tissue allowing the lung to push across the mediastinum to the opposite side. (See Vol. I, p. 442 and p. 469.) Sudden rupture of a rigid mediastinum from excessive pressure may cause fatal shock, and conversely excessive pressure on a slack mediastinum may cause death from kinking of the inferior vena cava.

The extrinsic causes of cardiac displacement may be conveniently grouped under three headings, viz. : 1, displacement due to abnormalities of the bony thorax ; 2, displacement due to pulmonary diseases ; 3, displacement due to pathological alterations in the position of the diaphragm.

DISPLACEMENT DUE TO ABNORMALITIES OF THE BONY THORAX

Scoliosis with the Convexity to the Right is the commonest deformity of the bony thorax. Even slight degrees of scoliosis of this type result in displace-

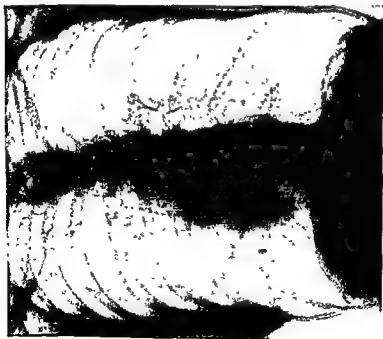


FIG. 7 Displacement of the heart to the right due to scoliosis with the convexity to the left.



FIG. 8—Severe kypho scoliosis without cardiac displacement.

ment of the heart to the left. Radiograms show the greater part of the heart shadow on the left side, the aortic knuckle is more prominent than usual, and the pulmonary artery shadow is very pronounced, the protuberance of the latter vessel causing the appearance of "mitralisation." In severe degrees of scoliosis the diaphragm may be very high and the apex of the heart hidden well below it. The sternum is usually deviated to the left side, and its border may be so superimposed on the aorta that it can simulate atheromatous plaques in the arch.

Scoliosis with the Convexity to the Left is rare. In mild deformities of this type the heart shadow lies more in the middle line and the vascular shadow appears a little wider than the average. In severe deformities the greater part of the heart shadow may lie to the right of the middle line and give an appearance simulating dextrocardia (Fig. 7). The trachea, however, is a useful guide to the presence of this deformity, as it swings very easily to the right side and always accompanies the heart and aorta if they are rotated or displaced to the right side.

Kyphosis causes varying types of cardiac displacement, dependent on the form of the scoliosis which nearly always accompanies kyphosis. In uncomplicated kyphosis the heart shadow appears small and the vascular shadow wide. In severe deformities there is an apparent hyperæmia due to compression of the lungs, but the pulmonary artery is not prominent.

DISPLACEMENT DUE TO PULMONARY DISEASES

Fibroid Phthisis is the commonest pulmonary disease displacing the heart. Fibrosis of the left upper lobe pulls the aorta and pulmonary artery to the left side, but seldom disturbs the position of the heart itself. Fibrosis of the left lower lobe pulls the heart to the left, but does not interfere with the aorta or pulmonary artery. Fibrosis of the whole left lung pulls the whole of the cardio-vascular shadow to the left side, and this displacement is aggravated by a compensatory emphysema of the healthy lung (Fig. 9). Right upper lobe fibrosis pulls the aorta and the superior vena cava to the right. Right lower lobe fibrosis pulls the heart to the right, but the ascending aorta and superior vena cava are not affected. Fibrosis of the whole right lung may result in the whole cardio-vascular shadow being displaced well to the right of the middle line. A useful guide to the degree of upper lobe fibrosis is the position of the main branches of the pulmonary artery. The left main branch is always higher than the right, usually by about half an inch. Upper lobe fibrosis practically always pulls the main branch of the pulmonary artery upwards, and the minor lower lobe branches are then seen running obliquely downwards, and outwards instead of curving inwards and outwards. Either main branch may be pulled as high as the second interspace anteriorly.

Pleurisy with Effusion is also a common cause of cardio-vascular displacement. The degree of cardiac displacement is not a reliable guide to the quantity of fluid present. In individuals with a rigid mediastinum the heart

rotates instead of moving laterally, and there may be little or no apparent alteration in its position. The trachea at the level of the 3rd to 4th dorsal vertebrae is a better guide to the quantity of fluid, as this is the weakest point

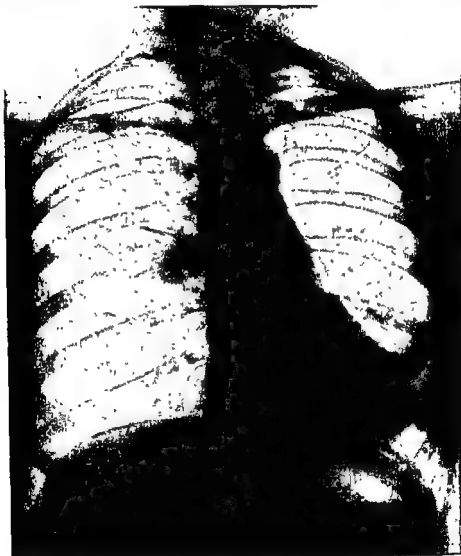


FIG 9—Displacement and rotation of the heart to the left due to fibroid phthisis and mediastinal pleuritis

in the mediastinum and the maximum effects of pressure are first noticeable there. Adhesions between the mediastinal leaves of the pleura sometimes follows pleurisy and pneumonia and may cause permanent displacement of the

heart. This appearance can be very misleading, as the lungs and spine are normal and the radiogram shows no apparent cause for the displacement (Fig. 9).

Callapse of the Lung

Collapse of a lower lobe usually produces slight displacement of the heart to the affected side. Collapse of the whole of a lung causes considerable displacement of the heart to the affected side, and the heart shadow may be invisible in the de-aerated hemithorax. The raised position of the diaphragm which accompanies total collapse of a lung is an obvious guide to the correct diagnosis.

Tumours

Primary tumours of the lung do not displace the heart shadow. Large neurofibromas arising from the intercostal nerves or large chondromas arising from an intervertebral disc may cause considerable displacement of the heart. Dermoid cysts of the superior mediastinum are usually situated anteriorly and often do not affect the position of the heart or great vessels. Dermoid cysts in the inferior mediastinum are usually intimately connected with the pericardium and cause much cardiac displacement. Substernal thyroid, if large, depresses the aorta and causes the vascular shadow to become wider, and the aortic knuckle to be lower and jutting out more to the left side.

DISPLACEMENT OF THE HEART DUE TO PATHOLOGICAL ALTERATIONS IN THE POSITION OF THE DIAPHRAGM

As already mentioned, the diaphragm plays a very large part in determining the shape and position of the normal heart. It follows that alterations in the position of the diaphragm will have a marked influence on the appearances of the normal heart. Some authors have paid undue attention to the presence of a large gas bubble in the stomach causing slight elevation of the left diaphragm and slight widening or rotation of the left cardiac border. It is often inferred on doubtful evidence that gaseous distension of the stomach is responsible for disturbance of the cardiac rhythm or præcordial discomfort. In a series of 1,000 normal individuals examined clinically and by X-rays in Westminster Hospital it was noted that all individuals with a transverse type of heart and rather high left diaphragm had a large gastric gas bubble. In none of these individuals were there any signs or symptoms of functional or organic disease of the heart. This large gas bubble is part of the normal habitus, and it persists throughout the life of the individual. There is no evidence to prove that a large gastric gas bubble can influence the cardiac function, and in the examination of a cardiac case its presence should be ignored.

Diaphragmatic Hernia

Diaphragmatic hernia of the gross congenital type, allowing the abdominal viscera to enter the thoracic cavity, causes gross displacement of the heart to



FIG. 11 —Displacement of the heart to the left due to paralysis of the right diaphragm.



FIG. 10 —Displacement of the heart to the right due to exentration of the left diaphragm.

the side opposite to the hernia. The small acquired hiatus herniæ, cases of short œsophagus, and cases with a defect in the anterior part of the diaphragm do not materially affect the position of the heart as seen in the postero-anterior view. In the two former lesions the stomach is behind the heart, and in the latter lesion it is in front of the heart.

Eventration of the Diaphragm, a rather rarer condition than diaphragmatic hernia, is usually seen on the left side and often causes such marked displacement of the heart to the right that clinically dextrocardia is diagnosed (Fig. 10). In a small number of cases of eventration the heart is not displaced, and the left border is beautifully seen through the raised diaphragm. The same state of affairs can be noted when the left diaphragm is artificially paralysed for therapeutic purposes. In some cases there is considerable cardiac displacement and in some no obvious displacement. Rigidity of the mediastinum probably accounts for the absence of displacement in some cases.

Emphysema is the commonest intrapulmonary disease causing depression of the diaphragm. Generally speaking, we see an apparently small heart in emphysema. The transverse diameter appears small relative to the transverse diameter of the chest. The two factors producing this apparent decrease in size are : (a) the actual increase in the antero-posterior diameter of the chest ; and (b) as the diaphragm is depressed by the over-distended lungs the heart rotates to the middle line. The question of enlargement of the right heart in emphysema is a complicated one and is discussed in detail in the section on enlargement.

Pleurisy with Effusion usually depresses the diaphragm on the affected side, but, as the fluid causes considerable lateral displacement of the heart, the effect of the depressed diaphragm does not come into play.

Pregnancy

Elevation of the diaphragm is produced physiologically by the pregnant uterus between the fifth and ninth months. In the pregnant woman the heart is usually of the transverse type during the last few months. Orthodiagrammatic measurements will, however, show the heart to be within the normal limits, and the heart-lung coefficient maintains its normal ratio between 1 to 1.9 or 2. A woman with an oblique or even narrow type of heart will show the transverse type in the last month of pregnancy. This alteration is largely due to elevation of the diaphragm, but partly due to the altered position of the spine in the pregnant woman.

Ascites and Large Intra-abdominal Tumours which elevate the diaphragm cause the heart to become more transverse in type. There is little likelihood of errors being made in these cases, as the transverse diameter of the chest also widens as the diaphragm rises, and the heart-lung coefficient is maintained within normal limits.

CHAPTER IV

DILATATION AND HYPERTROPHY OF THE HEART

RADIOLOGY HAS thrown much light on the nature of cardiac enlargement, and all interested in the subject should study *Parkinson's* Lumleian lectures. There are no radiological appearances which allow of a differentiation between dilatation and hypertrophy. An hypertrophied heart may show measurements well within the normal limits. It is true that as the left ventricle hypertrophies, it rotates and the apex of the heart may be lifted above the diaphragm, but there are other non-cardiac conditions which may cause this; hence its value as a sign of hypertrophy is doubtful. The radiologist, in estimating the size of a heart, should concern himself solely with the question of enlargement, and, remembering that enlargement is a vital factor in determining treatment and prognosis, he should take the greatest care that all his technical factors are accurate.

Physiological enlargement of the heart, great enough to be measured on teleradiograms, occurs after an extra systole which fails to eject the usual amount of blood from the ventricle, or during Muller's experiment of forced inspiration. As already described, this physiological enlargement is much greater in children than in adults. The majority of workers do not believe in the possibility of sudden acute dilatation of the normal heart, and *Parkinson* suggests abandoning the term "acute dilatation" completely. There is definite proof that the heart does not dilate following violent exercise; it decreases slightly or maintains its normal size. In observing enlargement of the heart during pathological conditions *Parkinson* states that "it is rather the length of time taken to produce demonstrable enlargement than the rapidity with which it occurs that has impressed him." On the other hand, there are many isolated reports of cases of acute dilatation of the heart, and in some of these there was such rapidity in increase and decrease in size that pericardial effusion could be excluded as the causation of the sudden enlargement. *Roesler* believes that acute dilatation occasionally occurs, and cites as examples cases of coronary thrombosis and diphtheria. *Dorner*, during a severe epidemic of diphtheria, made numerous radiological observations and found gross enlargement of the heart in severe cases on the third day of the disease. The author found considerable enlargement of the heart in a young doctor three or four hours after an attack of coronary thrombosis. There had been no previous record of the size of the heart in this patient, but, three months after the attack, teleradiograms showed the heart to be well within the normal limits. A more striking instance of rapid cardiac enlargement was

observed in a young woman of 25, whose chest was X-rayed to determine the cause of pyrexia following an abdominal operation. The first pictures showed a bilateral bronchiectasis with a superimposed pneumonitis on the right side. The heart was within the normal limits. Radiograms taken six days later showed the heart shadow to have increased in size by about $1\frac{1}{2}$ cm. At the time of the second examination there was a profound anæmia (2,500,000 R.B.C.). A transfusion was given on the following day, and two days later radiograms showed the heart again within the normal limits. There was some ground for believing that the anæmia was caused by injections of sulphanilamide, but whether this drug, the anæmia, or the general toxic condition was responsible there was no doubt but that a considerable increase and decrease in the size of the heart had taken place over a period of ten days. The pulsation of the heart at the time of the second examination was forcible enough to exclude the possibility of a pericardial effusion.

In all cases of cardiac enlargement, the heart should be examined with a view to determining which chambers are chiefly affected.

CONDITIONS CAUSING CARDIAC ENLARGEMENT

Hypertension

This is one of the commonest causes of general enlargement, although the left ventricle is the chamber chiefly affected. The degree of enlargement is dependent on many factors. In elderly people with long-standing hypertension, the heart is enlarged to the left and backwards and the apex is often raised above the diaphragm. In cases with a rigid superior mediastinum, the enlarged left ventricle may displace the right cardiac border to the right. The aortic knuckle is usually slightly dilated and projects more to the left side. In arteriosclerotic cases the aorta is unfolded and higher than normal, and plaques may be visible in it. The indentation between the aortic knuckle and the left ventricle is never so pronounced as it is in aortic incompetence, and, generally speaking, one can describe the heart in hypertension as being rather of the squat type. If the pulmonary artery is enlarged and the configuration of the heart becomes of the mitral type, this is evidence of beginning failure. The predominance of the aortic knuckle in this stage of hypertension is a useful point in excluding mitral disease. In rare cases prolonged hypertension may have been present without any radiographic evidence of abnormality of the cardio-vascular shadow. It is possible that these are cases in which the heart to begin with was of the small narrow type. In malignant hypertension or secondary renal hypertension in young people the aortic shadow is always dilated. *Roesler* shows that this is a dynamic dilatation due to increase in the internal pressure. In cases of this nature autopsy shows the aorta to be normal.

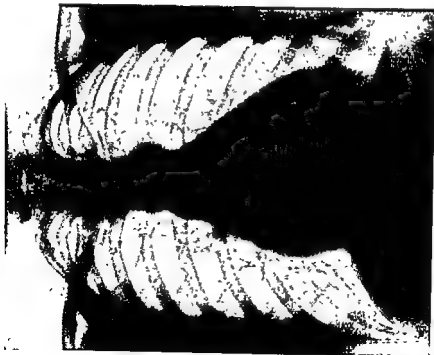


FIG. 12.—Enlargement of the heart in hypertension.
Aorta normal.



FIG. 13 — Enlargement of the heart to the left in hypertension
with generalised arteriosclerosis. Aorta unfolded.

Renal Diseases

In the early stages of acute glomerulo-nephritis there is usually considerable enlargement of the heart, but as this condition is frequently associated with œdema of the lungs, it is probable that hydropericardium plays a considerable part in the enlargement of the shadow. In chronic interstitial nephritis there



FIG. 14.—Enlargement of the heart to the left in hypertension.
Aorta normal.

is usually enlargement of the left ventricle ; the right border of the heart, the aorta, the pulmonary artery, and conus pulmonalis appear normal.

Hypotension

Sudden drop in blood-pressure, such as occurs during fainting, is accompanied by a measurable increase in the transverse diameter of the heart shadow. This enlargement is probably due to bradycardia, *Roesler* having shown that bradycardia alone is sufficient to cause enlargement of the heart shadow. *Roesler* also noted, in two cases of sudden drop in pressure following moderate exertion, that there was no increase in the size of the heart, but a decrease in the size of the aortic shadow.

Rheumatic Carditis

Parkinson discusses a number of reported cases of rheumatic carditis without endocarditis. This condition is obviously very rare and is probably associated with considerable enlargement of the heart. From the few cases reported it is impossible to determine whether the enlargement might not have been a result of previous attacks. *Parkinson* points out that there is no radiographic evidence that the average patient with a first attack of rheumatism with the heart affected does, in fact, show early enlargement from acute myocarditis.

Diphtheria

Dorner has shown that in very severe cases there may be considerable enlargement of the heart shadow as early as the third day of the infection. In less severe cases, showing evidence of cardiac involvement, enlargement was not noted until the second or third week. In some cases a certain degree of enlargement persists, but with suitable treatment the heart usually returns to its normal size after a few weeks or months. With modern methods of treatment and prevention, diphtheria is fortunately becoming rare, and there is not much likelihood of adequate material being forthcoming for more exhaustive radiological investigation.

Pneumonia

The evidence for or against radiological evidence of cardiac enlargement during pneumonia is indecisive. In the investigation carried out by *Davies*, *Hodgson*, and *Whitby*, three cases out of 119 showed X-ray evidence of cardiac enlargement, and in these three cases there was no clinical evidence of cardiac weakness. On the other hand, it seems probable that there must be some enlargement of the heart if extensive areas of lung tissue are consolidated. Alterations in the position of the heart due to consolidation of a large area of lung tissue, or to pleurisy with effusion, make estimations of the radiological size of the heart very inaccurate during pneumonia.

Myocarditis

Severe myocardial damage may be present without any X-ray evidence of abnormality of the cardio-vascular shadow. Most cases usually show enlargement of the heart to both sides and a characteristic "flickering" pulsation of the left ventricle. The heart also shows wide variations in size with the phases of respiration, and the main pulmonary vessels near the roots of the lungs are engorged.

The question of acute dilatation occurring with coronary thrombosis has already been discussed. In a series of 200 cases observed by *Parkinson*, he concluded that there were 128 with enlargement of the heart. He estimates

that, in the vast majority of these, hypertension was the only or predominant cause of the enlargement; but there were 11 (8.6 per cent.) in which he believed that cardiac infarction was the sole cause of the enlargement.

Hyperthyroidism

A considerable number of radiological investigations of the heart in thyrotoxicosis have been published. In severe cases there are certain characteristic

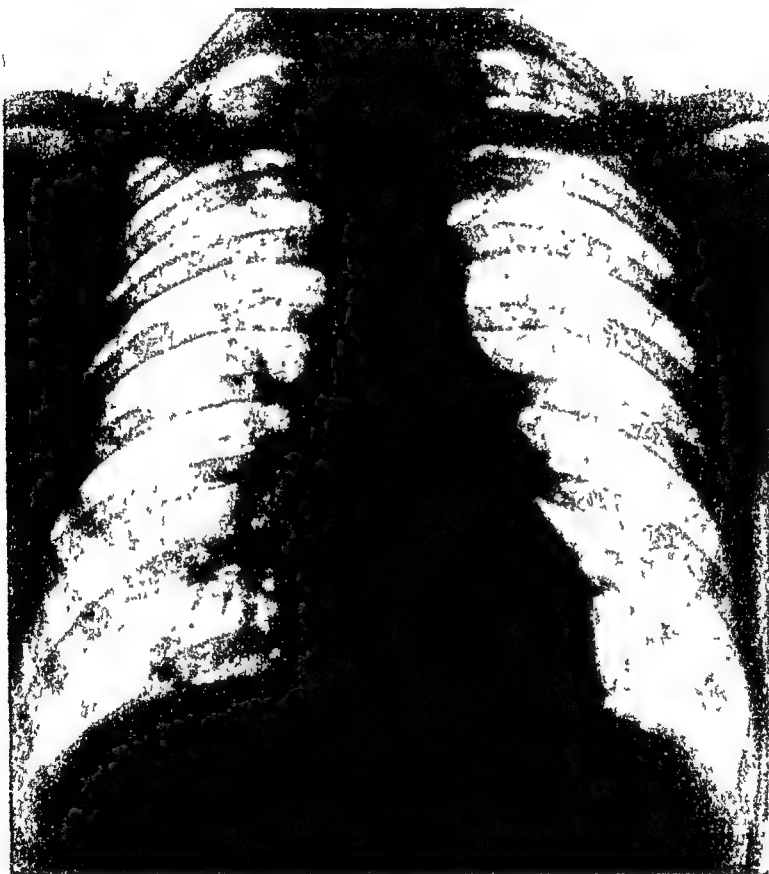


FIG. 15.—Thyrototoxic heart. No enlargement of the transverse diameter but considerable swelling of the pulmonary artery.

features. There is some enlargement of all the chambers of the heart; the pulmonary artery and conus pulmonalis on the left side are increased in size, giving an impression of mitral disease; and the superior vena cava shadow on the right side is usually very pronounced. Practically all workers have commented on the curious pulsation in thyrotoxicosis. The pulsation is more obvious on the vascular than the cardiac shadow, and on the screen the movements give the impression of a sponge being squeezed in and out: hence

the term "sponge heart." The heart shadow and the pulsation may return to normal after successful treatment or some degree of enlargement may persist.

Myxœdema

Mild cases show no change in the heart shadow. Severe cases, according to *Roesler*, show enlargement to both sides with the flask-like shape of pericardial effusion. *Roesler* states that the enlargement is due to cardiac dilatation, probably associated with pericardial effusion. Despite the bradycardia the pulsation is sluggish and of diminished amplitude. *Palmer* describes a case in which there was slight general enlargement of the heart with gradual clinical improvement and return of the heart to its normal size after thyroid medication.

Deficiency Diseases

In severe cases of rickets and scurvy cardiac enlargement has been noted. A classical example of general cardiac enlargement due to chronic avitaminosis has been reported by *Rabinovitz* and *Rogers*. In beriberi *Roesler* describes globular enlargement of the heart and the pulmonary artery or coarctation of the pulmonary artery. The superior vena cava is dilated and there is vigorous pulsation of the conus. A pericardial effusion may be present. A striking feature is the absence of X-ray evidence of congestive lung failure, despite clinical evidence of right heart failure. Deficiency diseases are often associated with a severe anaemia, and this may be an important factor in the production of cardiac enlargement.

Idiopathic Heart Hypertrophy

In recent years an increasing number of cases of cor bovinum in infants have been recorded. Radiograms reveal enormous enlargement of the heart to both sides. In a few cases there was a coexistent enlarged thymus. Autopsies have not clarified the aetiology. In some cases there was nothing found but pure hypertrophy of the heart muscle, and in others nests of lymphocytes were embedded between the hypertrophied muscle fibres. *Roesler* holds that many of these were cases of glycogen storage disease.

Anæmia

Most cases of severe anaemia, irrespective of its nature, show some degree of general enlargement of the heart. The exact causation of this enlargement is quite unknown, but if the blood picture is restored to normal the heart returns to its normal size. The author found enlargement of the heart to the extent of 1 and 1.5 cms. in two women suffering from microcytic anaemia. It is estimated from the histories that the anaemia had been present for 10 and 15 years respectively.

Phosphorus, Alcohol, and Coal-gas Poisoning

These toxins are said to cause general enlargement of the heart shadow.

Arterio-venous Aneurysm

This condition is usually traumatic in origin, and many cases were observed during the Great War. If a fistula develops between two vessels of large calibre, the heart enlarges. The enlargement may be general, or may be limited to the right heart, and there is considerable passive hyperæmia of the lung vessels. The mechanism causing the cardiac enlargement is not quite clear. *Dean* shows that one of the principal factors is the shunting of a large amount of arterial blood back to the heart, but he also believes that lowered peripheral resistance plays a part. If the shunt is temporarily interrupted by external pressure, the heart decreases in size, and after successful operative treatment of the anastomosis the heart returns to normal.

Deformities of the Bony Thorax

The displacements of the heart due to scoliosis have already been discussed. With severe degrees of kypho-scoliosis remarkable alterations in the shape of the heart can occur, and the aorta may be kinked. It is doubtful, however, if displacement of the heart and aorta, no matter how severe, can be a primary cause of cardiac enlargement. Some very large hearts have been described in hunchbacks, but other factors than the bony deformity were probably responsible for the enlargement.

Trauma of the Heart

Comparatively few radiological records of heart wounds were made during the Great War. There is, however, a considerable literature in America on penetrating and non-penetrating wounds of the heart. In most cardiac wounds there is a rapid pericardial hæmorrhage, and raised intrapericardial pressure is the commonest cause of death in these cases. It is unwise in most cases to waste time making an X-ray examination, as the diagnosis of blood in the pericardium can be made more promptly by aspiration. In cases, however, where an opaque foreign body, such as a bullet, has lodged in the heart, X-ray examination is necessary to locate this. If the bullet is in the ventricle it shows a dancing movement on the screen and changes in position with changes in posture. If the bullet is in the myocardium it shows lateral pulsation and does not alter in position with posture. If the bullet is in the pericardial sac it changes in position with changes in the patient's posture unless it is held in the one place by adhesions or organisation of blood. In the vast majority of cases radiography shows gross enlargement of the cardiac

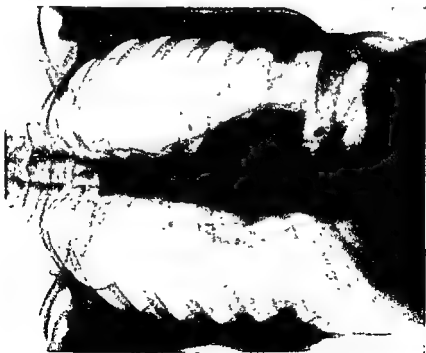


Fig 16.—*Erect radiogram.* Bullet in the ventricle: Professor Roesler is of the opinion that this bullet was in the right ventricle.



Fig. 17—Same case as Fig. 16 taken in the supine position.

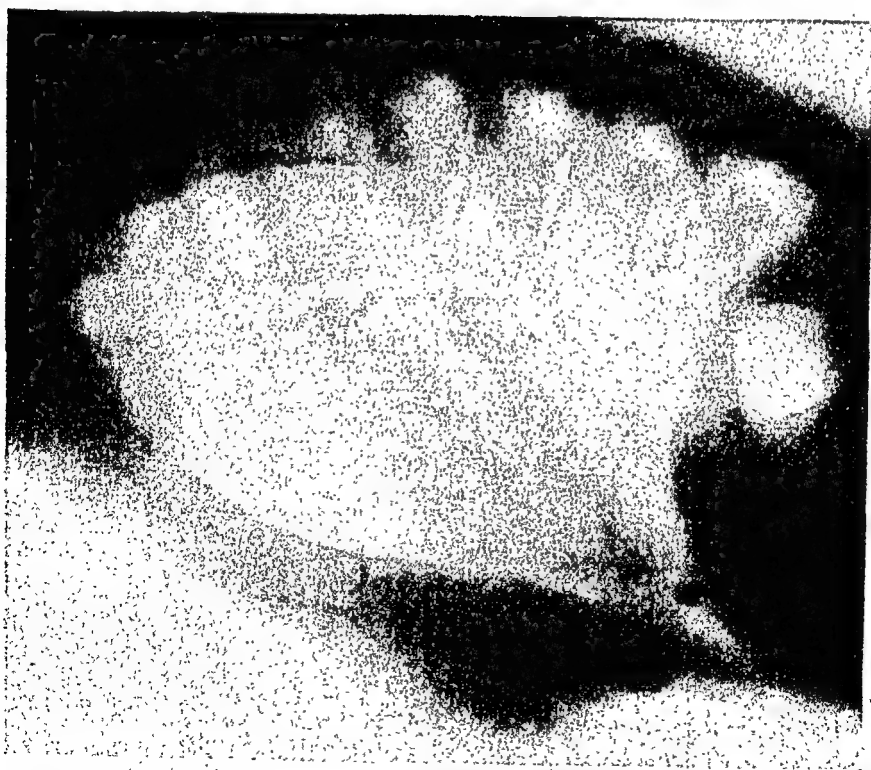


FIG. 18.—Same case as Fig. 16. Lateral view erect.

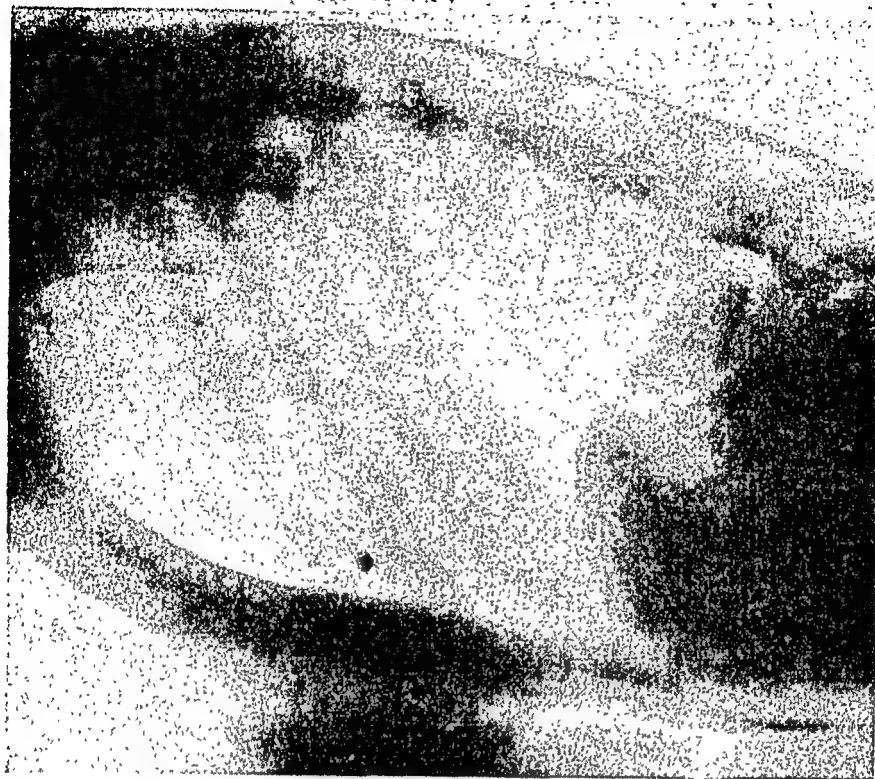


FIG. 19.—Same case as Fig. 16. Lateral view supine.

shadow, this being chiefly caused by blood in the pericardium. In a case seen by the author in Westminster Hospital, there was no enlargement of the heart. The patient was a young girl, shot at close range with a .22-mm. revolver. There were three wounds on the left side of the thorax. X-ray examination twenty-four hours after the wounding showed two of the bullets lying in the soft tissues of the chest wall and a third superimposed on the shadow of the ventricles. Screen examination and radiography in different postures showed that the bullet was actually in one of the ventricles. There was no enlargement of the heart shadow. The patient made an uneventful recovery. *Dr. Roesler* examined the pictures and concluded that the bullet was probably in the right ventricle. The fate of a bullet in the ventricle is variable: it may become jammed in the chordæ tendineæ or it may be shot into the circulation and find a permanent site in a vessel far distant from the heart. For this reason it is imperative to radiograph an intracardiac foreign body immediately before its attempted removal.

Pulmonary Disease

There is a general impression that right heart enlargement and failure ensue as a result of chronic pulmonary disease with obliteration of large areas of the vascular bed of the lungs. This impression is not borne out by radiographic investigations, which usually show an apparent small heart in cases of asthma, emphysema, chronic phthisis, and pneumoconiosis. There are many degrees and varieties of *emphysema*, and the only safe criterion on which its extent can be estimated is the X-ray demonstration of the absence of vascular markings in an area of increased translucency. Emphysema is never a primary condition, and by itself it is doubtful if it can cause gross enlargement of the right heart. Actually, in emphysema, blood is returning to the right heart, not only more slowly, but in diminished quantities, and the result is a real diminution in the size of the heart. This diminution in size is enhanced by the effect of depression of the diaphragm and widening of the transverse and antero-posterior diameters of the chest. *Parkinson* goes into this question very carefully and points out that enlargement of the right ventricle manifests itself chiefly by an enlargement of the conus pulmonalis, best seen in the second oblique position. This enlargement of the conus may, however, be dynamic, a theory which is supported by *Rubin's* findings. *Rubin* in a group of selected cases found no evidence of right heart enlargement in emphysema. In the author's opinion, if considerable cardiac enlargement and emphysema are found together, it is more than likely that some cause other than the emphysema is responsible for the enlargement.¹

¹ In a more recent publication, *Parkinson* and *Hoyle* describe the cardiac changes in eighty cases of emphysema. They conclusively show that in 40 per cent of cases there is a true enlargement of the conus pulmonalis and in about half of these the body of the right ventricle was also enlarged. In about 30 per cent of cases the left ventricle was enlarged, but this was due to co-existent systemic hypertension or, in a few cases, to coronary disease.

There is little or no mention in the literature of enlargement of the heart in association with *carcinoma of the lungs*; yet in the author's experience at the Royal Chest Hospital, pulmonary carcinoma is more often associated with gross cardiac enlargement than any other pulmonary disease. The enlargement may be general or limited to the right heart and is due to one or more of the

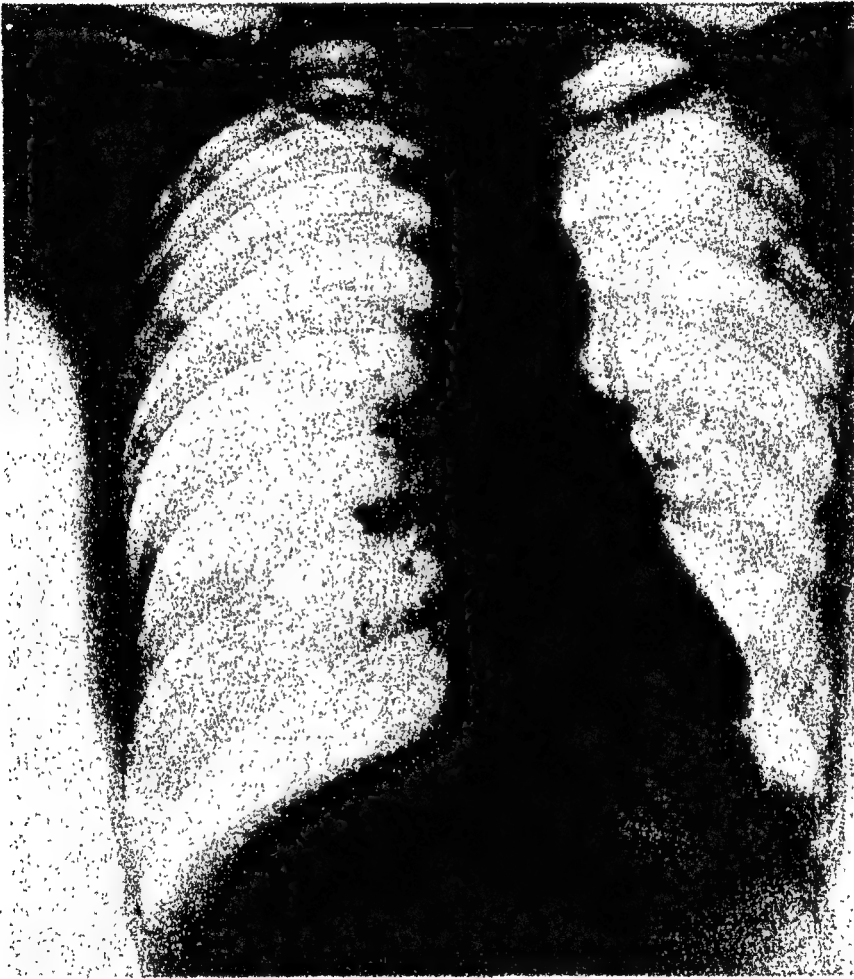


FIG. 20.—Enlargement of the *conus pulmonalis* in a case of emphysema.

following factors: (1) Malignant infiltration of the superior vena cava, often associated with generalised malignant infiltration of the lungs. (2) Malignant infiltration of the pericardium with or without fluid in the pericardial sac. (3) Constriction of the pulmonary artery by the growth. In one case of extensive malignant infiltration of the myocardium serial radiograms showed no alteration in the size of the heart, but this heart was already enlarged from hypertension.

Obesity

Fatty infiltration of the heart is rare as a primary cause of cardiac enlargement or failure. The heart in obese individuals is always of the squat transverse type, due to the diaphragm being raised by increased intra-abdominal fat. The aorta is cramped and rotated, so that both ascending and descending parts of the arch lie clear of the vertebral shadow. There is invariably an accumulation of fat in the pleuro-pericardial angle on the left side, and this is not infrequently mistaken for the left border of the heart. Careful screen examination and hard radiograms will show the true nature of this opacity. Obese individuals also show a large gastric gas bubble; this is a normal finding and should never be regarded as a cause of cardiac symptoms. If a very large heart is found in an obese individual it is probable that there is an additional factor, such as hypertension or coronary disease, causing the enlargement.

Cardiac Aneurysm

Cardiac aneurysms are now recognised as being comparatively common, the causative factor being coronary arteriosclerosis and thrombosis. Such aneurysms are usually found near the apex of the heart. Probably 5 to 10 per cent. of heart aneurysms can be detected radiologically. The aneurysm in its early stages may not be visible as a bulge on the left border, but can be identified by a characteristic square shape of the left border of the heart with absence of pulsation, or flickering pulsation, over the affected area. In long-standing cases a true aneurysmal bulge can be seen on the left border, and there are a few records of visible calcification in the wall of the aneurysm.

Not infrequently two cardiac aneurysms may be present in the one individual. In most cases of cardiac aneurysm, X-ray examination shows nothing but general enlargement of the heart, the aneurysm being invisible because it is situated in such a position that it cannot be brought into profile or because there are pericardial adhesions over the affected area.

Primary Tumours of the Heart

These are rare, and usually affect the auricles. Angiosarcoma and rhabdomyoma of the auricles have been described. *Ehrenberg* has recorded a case of sarcoma of the right auricle with a bulging of the auricle to the right and swelling of the superior vena cava. *Kause* described a similar tumour of the left auricular appendage, which appeared as a semicircular opacity arising from the upper part of the left cardiac border and pulsating synchronously with the aorta.

Disorders of Rhythm and Conduction

Paroxysmal Tachycardia.—This condition usually causes no alteration in the radiological size of the heart. If it persists for a long time it may cause

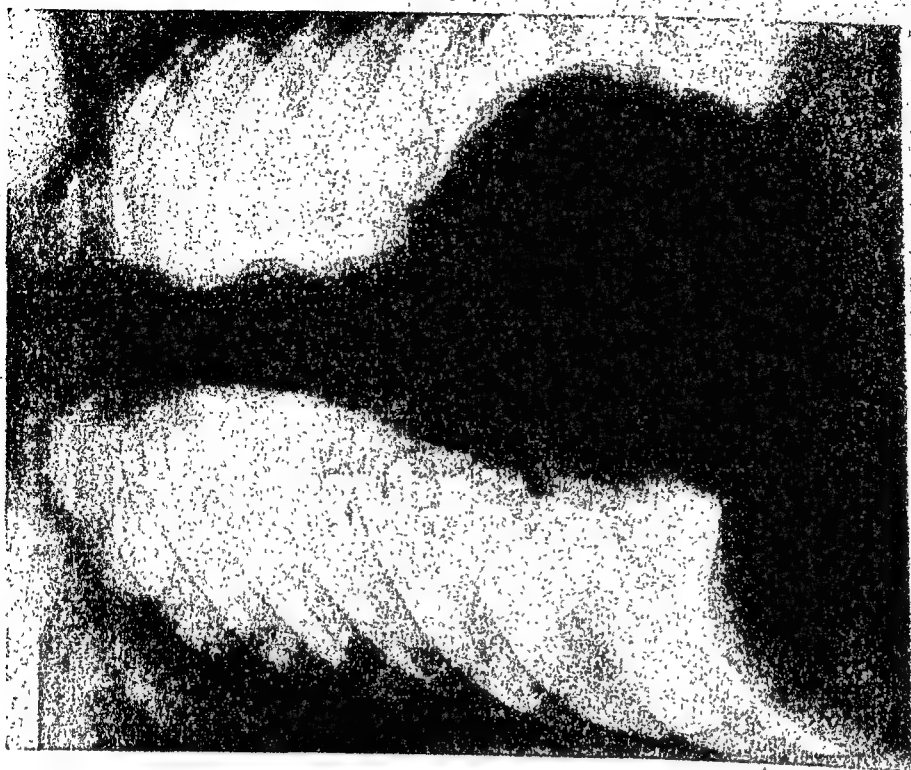


FIG. 22.—Metastatic sarcoma in the heart. E.C.C. normal. Autopsy revealed extensive infiltration of the left auricle and left ventricle. Primary tumour removed from the orbit 10 years previously.

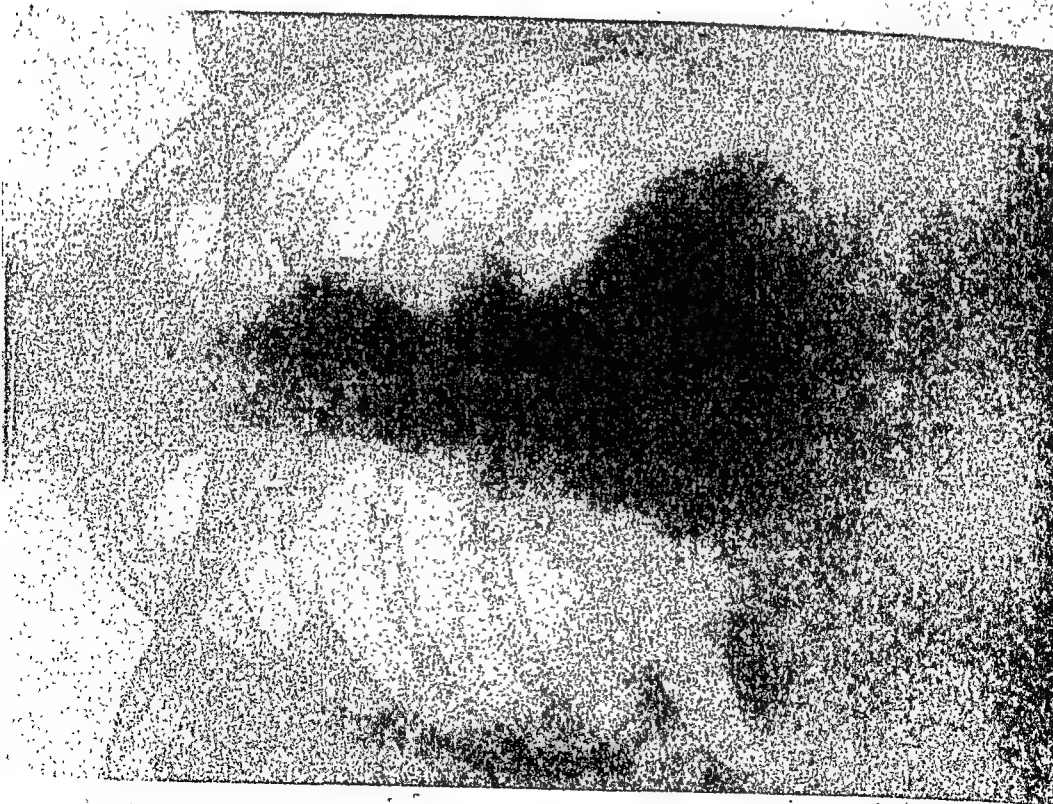


FIG. 21.—Chordal aneurysm following two attacks of coronary thrombosis. Note the "squire" shape of the heart and the small bulge just below the left diaphragm.

congestive failure, which rapidly disappears when the tachycardia is controlled. *Palmer* describes a patient, aged 62, who had severe and frequent attacks of paroxysmal tachycardia for 45 years, and who showed no radiological evidence of cardiac enlargement.

Auricular Fibrillation and Auricular Flutter, if unassociated with organic heart disease, may exist for many years without causing cardiac enlargement.

Bradycardia usually causes enlargement, but as *Roesler* points out it is the diastolic size which surpasses the normal standard. The author observed in a young girl, with a heart rate of 54, enlargement of both the systolic and diastolic size.

Complete heart block appears to be associated with enlargement in practically all cases. Partial heart block is not necessarily associated with enlargement.

Congestive Heart Failure

The question as to whether the heart enlarges or not during an attack of congestive failure is still unsolved. In most cases the heart is already enlarged before failure begins. The engorgement of the liver and the diminished air content of the lungs combine to raise the diaphragm to an abnormally high level. The elevation of the diaphragm flattens the heart out so that there is apparent enlargement both to right and left. Distension of the right auricle also adds to the impression of enlargement and in many cases there is probably a small undetectable pericardial effusion present. The vascular shadow may also be flattened out and appear wider than normal, an effect which is enhanced by engorgement of the superior vena cava. The pulmonary conus is always dilated, causing the so-called "mitralisation" of the heart.

Hydrothorax is a frequent complication of failure, and is usually first seen on the right side. A small hydrothorax is easily overlooked unless the patient is screened or radiographed in oblique positions. Even a large hydrothorax may not result in displacement of the heart to the opposite side: the deflation and loss of elasticity of the engorged lung more than counterbalances the pressure effect of fluid in the pleural cavity.

CHAPTER V

VALVULAR DISEASES OF THE HEART

MITRAL STENOSIS

THIS is the commonest valvular lesion of the heart and in chronic cases gives typical X-ray findings. In the diagnosis of mitral stenosis the X-ray demonstration of an enlarged left auricle ranks in importance with the presystolic murmur. The normal left auricle is situated posteriorly, and in the routine postero-anterior view only its appendage forms part of the left cardiac border. In fact, the widest part of the normal left auricle is central, or even situated a little to the right of the middle line. In mitral stenosis the chamber first and chiefly affected is the left auricle, but the shadow of the auricle is not the factor responsible for the so-called "mitralisation" of the heart. A mitralised heart is characterised by a small or absent aortic knuckle, and a prominent pulmonary artery and conus which usually merge without a break into the shadow of the left ventricle. As already pointed out, this mitral appearance may be normal or may be produced by many lesions which have nothing at all to do with the mitral valves. To be sure of the diagnosis of mitral stenosis the radiologist must demonstrate hypertrophy or dilatation, or both, of the left auricle.

In a chronic case of mitral stenosis the left auricle can be clearly visualised in the postero-anterior and oblique views. In the postero-anterior view the enlarged auricle is seen as a dense pear-shaped opacity inside the normal cardiac shadow (Fig. 23). The apex of the pear is just below the pulmonary artery on the left border and the rounded base of the pear is superimposed on the upper part of the right auricle on the right border. In the first oblique position the enlarged auricle projects into the posterior mediastinal space and renders opaque a normally translucent space, but as the posterior mediastinal space can also be obliterated by general enlargement of the heart a barium swallow is necessary to differentiate between the two conditions. In the vast majority of cases of mitral stenosis the enlarged left auricle displaces the œsophagus to the right side (Fig. 27). This displacement is best noted in the *first oblique position*. On watching the barium, one notices a slight delay at the level of the bifurcation of the trachea and then an abrupt deviation of the bolus in the œsophagus to the right. The delay and the abruptness of the deviation are important, as in general enlargement of the heart the œsophagus is sometimes slightly deviated to the right; but this deviation is gradual and is not associated with a pause at the level of the bifurcation. There are reports of displacement of the œsophagus to the left by an enlarged

but this is rare. Not infrequently there are reports of dysphagia being a prominent symptom in mitral stenosis with œsophageal displacement. In the author's experience the œsophagus can be displaced several inches to either side



FIG. 23 Mitral stenosis with a severe degree of passive hyperæmia of the lungs. Note the dense opacity of the left auricle superimposed on the shadow of the right auricle

of the middle line without producing œsophageal symptoms, and it is likely that dysphagia with mitral stenosis is probably due more to varices in the œsophageal wall than to displacement of the œsophagus. This view is

supported by the comparative frequency of dysphagia in elderly people with congestive heart failure.

In many cases of mitral stenosis the enlarged left auricle pushes upwards

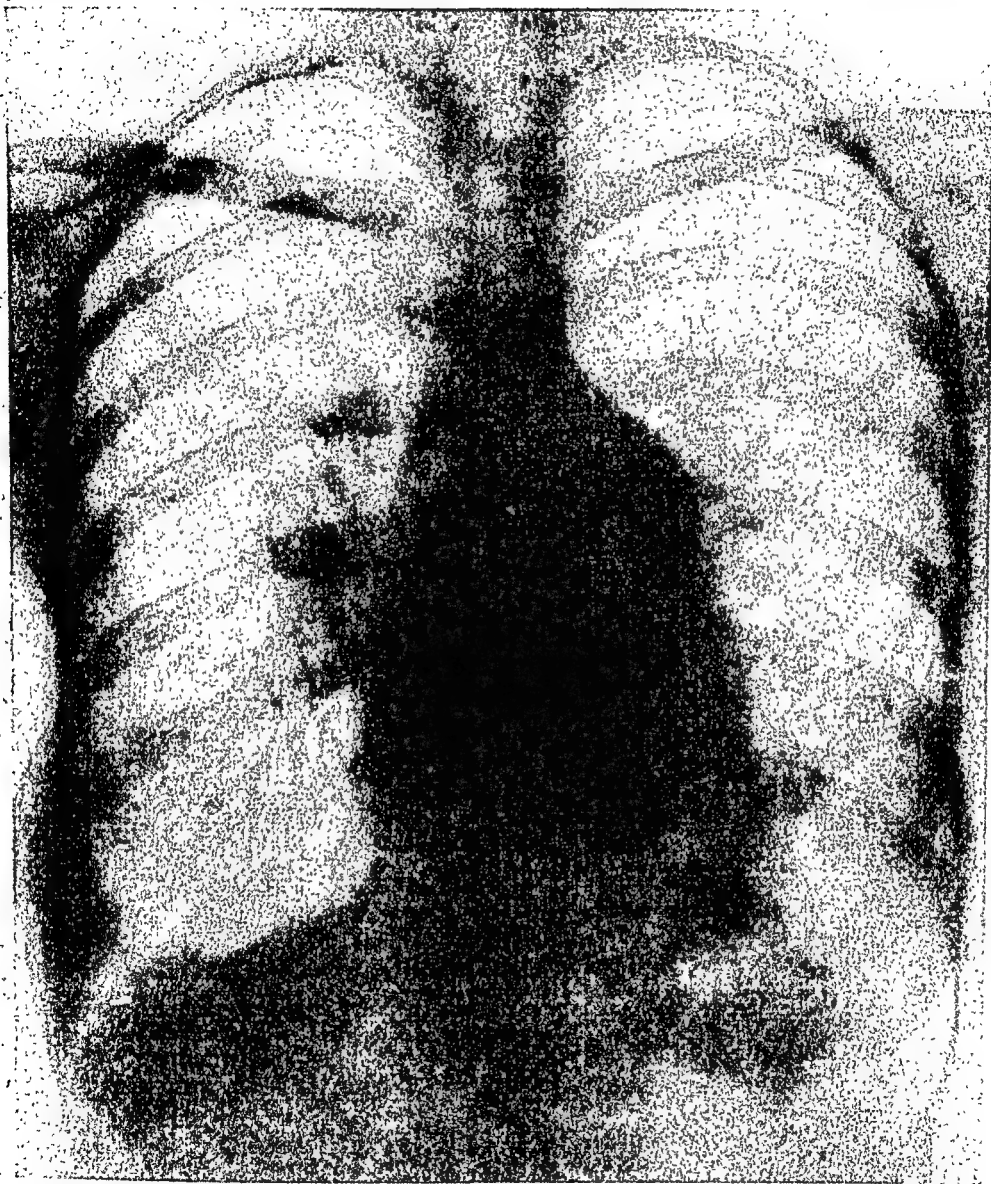


FIG. 24.—Mitral stenosis with passive hyperemia of the lungs. Note the dilated pulmonary artery and the hypoplasia of the aorta.

into the bifurcation of the trachea and actually causes a splaying of the bifurcation. This deformity is again best visualised in the first oblique view, which may clearly show the left main bronchus jammed against the pulmonary

artery (Fig. 28). Dynamic dilatation of the pulmonary artery is a frequent occurrence in mitral stenosis, and if this vessel does not yield, the left main bronchus may be narrowed as a result of pressure from below by the auricle and above by the artery. In rare cases the enlarged auricle rides so high into



FIG. 25. —Mitral stenosis. The *conus pulmonalis* is clearly visible as a separate bulge between the swollen pulmonary artery and the left ventricle. There is hypoplasia of the aorta.

the bifurcation that it pushes both left main bronchus and pulmonary artery hard into the base of the aorta, thereby squeezing the left recurrent nerve and causing left recurrent paralysis. *Parkinson* and *Bedford* were the first to point out the clinical significance of a small indentation on the oesophagus at

the level of the left main bronchus and pulmonary artery. They called this the pulmonary artery impression, a rather misleading term, as this artery is not in contact with the œsophagus. This indentation on the œsophagus is probably caused more by the left main bronchus than the pulmonary artery, but in mitral stenosis the dilated artery pressing on the left main bronchus causes an increase in the depth of this normal indentation. Summarising the œsophageal appearances in the first oblique view in mitral stenosis, we find the aortic indentation small or absent, the left bronchus indentation increased, and below this a wide deviation due to enlargement of the left auricle.

We must now consider in detail the appearances in the *postero-anterior view*. The shadow of the aortic knuckle is small. This smallness is real, as in mitral stenosis there is usually a true hypoplasia of the aorta, especially if the disease has developed early in life. The pulmonary artery and conus pulmonalis are usually prominent; this is a dynamic dilatation of the artery, as autopsies seldom show true dilatation of the pulmonary artery in mitral disease. The left ventricle may or may not be enlarged, depending on the stage of the disease; it is almost always rotated a little to the left and shows diminished pulsation. The lower right border is usually a little sharper defined than the average, and if the right ventricle is enlarged it may be pushed slightly outwards and to the right. The shadow of the superior vena cava is well marked, and in children the triangular shadow of the normal azygos vein lying in the angle formed by the vena cava and the right main branch of the pulmonary artery is very prominent. The whole appearance is dominated by the pear-shaped opacity of the enlarged left auricle visible through the heart shadow. The visibility of the left auricle in the postero-anterior view is largely dependent on an increase in the thickness of the wall of the auricle. A simple dilatation of the auricle would not result in any alteration of the normal densities of the heart shadow as seen in the postero-anterior view. This is a very important point, as in a number of cases of mitral stenosis the auricle hypertrophies long before it dilates. With good technique the hypertrophied auricle can be easily seen in the postero-anterior view, although oblique views may show no evidence of mitral disease and the course of the œsophagus to be normal. In these cases also the conus pulmonalis and pulmonary artery are not dilated and a superficial study of the heart shadow in the postero-anterior view may give the impression that the heart is normal.

In very severe cases the auricle, as it dilates, extends out over the right border of the heart and we get a double shadow to the right of the middle line, the upper shadow being formed by the left auricle and the lower shadow by the right auricle. Not infrequently in these cases ventricular pulsation is transmitted to the left auricle and see-saw pulsation is seen on the right border. Occasionally the left auricle enlarges to such an extent that it forms the whole of the right border of the heart, and we speak of aneurysmal dilatation of the left auricle (Fig. 29). The shape of the heart is then very similar to that seen in pericarditis

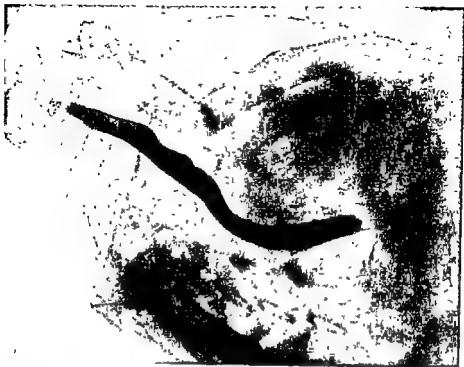


FIG. 27.—Mitral stenosis with displacement of the a-splingus backwards and to the right. First oblique view.



FIG. 28.—Mitral stenosis with prominent enlargement of the conus and a relatively small pulmonary artery.

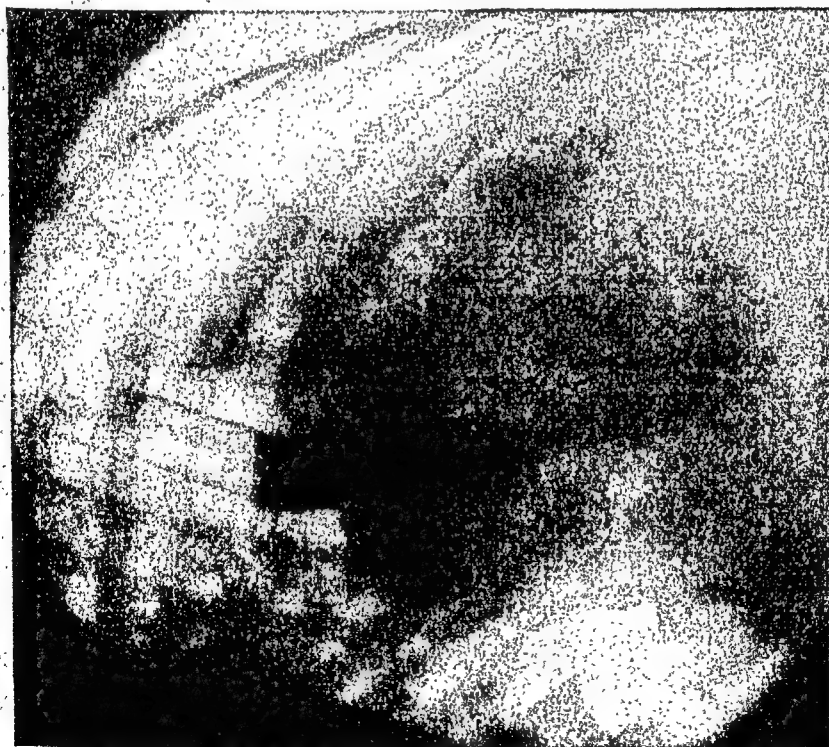


FIG. 28.—Mitral stenosis with displacement of the esophagus to the left. First oblique view.

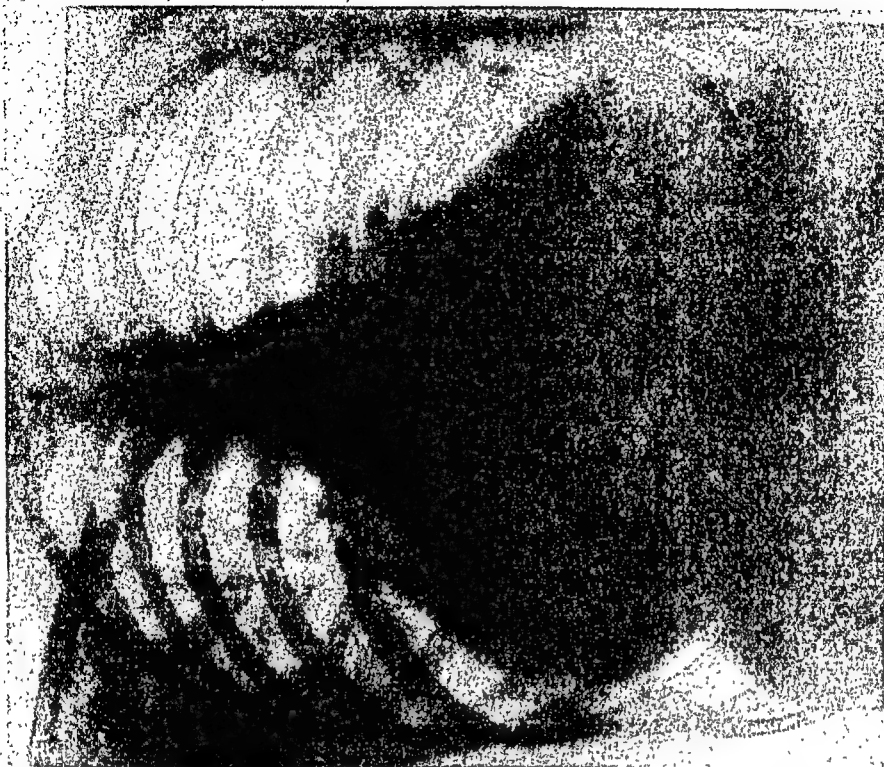


FIG. 29.—Aneurysmal dilatation of the left auricle. Diagnosis confirmed by autopsy.

with effusion, and the differential diagnosis by radiography may be impossible. In this advanced stage of the disease a pericardial effusion may be present.

All cases of mitral stenosis show sooner or later hyperæmia of the pulmonary



FIG 30 —Calcification of the aortic valves.

vessels. *Assman* suggests that early in mitral disease a distended pulmonary artery may squeeze the left upper lobe vein and cause hyperæmia localised to the left upper lobe. This appearance is quite different from that of true passive hyperæmia which develops symmetrically in both lungs. The appear-

ances of passive hyperæmia are of the greatest importance both from the point of view of diagnosis and prognosis, and are discussed under a separate heading.

AORTIC VALVULAR DISEASE

X-ray examination in cases of aortic valvular disease is not so decisive as it is in cases of mitral disease. We are able by radiology to define an aortic configuration of the heart, but this does not necessarily mean that the aortic valves are stenosed or incompetent. An aortic configuration of the heart is as follows. In the *postero-anterior view* the aortic knuckle is high and prominent and the descending part of the arch can usually be seen well to the left of the spine. The pulmonary artery and conus pulmonalis are small and the shadow of the left ventricle is denser and better defined than usual. The apex of the heart usually lies well above the diaphragm. On the right side the ascending part of the aorta projects out over the shadow of the superior vena cava. The right auricular shadow is unaltered or, if the left ventricle is hypertrophied and rotated, this right auricular shadow may move to the left and may be superimposed on the spine. In the *oblique views* the whole course of the aorta is seen with remarkable clarity, and the posterior mediastinal space is brilliantly illuminated. Careful search should be made for calcified plaques in the aorta or calcification of the aortic valves. The aortic configuration is frequently seen in elderly people with hypertension and arteriosclerosis. Points in favour of regurgitation through the aortic valves are an increase in the size of the left ventricle and rapid forcible pulsation on both ventricle and aorta. Points in favour of stenosis of the aortic valves are no increase in size of the ventricle and very slow and forcible pulsation on both ventricle and aorta. In all cases of aortic disease the aortic impression on the œsophagus is increased.

COMBINED MITRAL AND AORTIC DISEASE

The X-ray appearances of a combined valvular lesion are characteristic. The heart has the mitral configuration with a prominent conus and pulmonary artery, but in addition the aorta is unduly prominent and conspicuous and dominates the picture. The right lower border (right auricle) is pushed out to the right and fairly forcible pulsation is visible on both borders. The shadow of the left auricle can seldom be seen in the postero-anterior view, and there is little or no stasis visible in the pulmonary vessels. A barium swallow shows all four œsophageal impressions increased in depth. The heart in combined valvular lesions has been aptly described as being like a cottage loaf.

INTRACARDIAC CALCIFICATION

Calcification of the *mitral and aortic valves* can be demonstrated radiographically with good technique. Such calcification is comparatively common, and may be demonstrated in all positions. The calcified valves appear as elliptical or sabre-shaped dense areas showing a typical dancing movement on the screen. On a radiogram they are easily confused with calcification of the costal



FIG. 31.—Combined mitral and aortic disease.

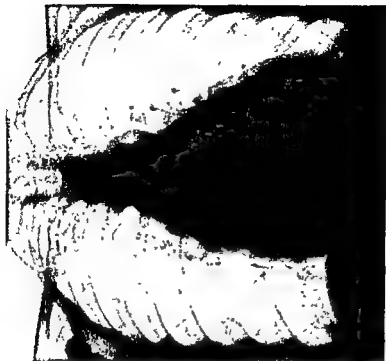


FIG. 32.—Combined mitral and aortic disease. Atheromatous plaques visible in the aorta

cartilages, and confirmation of the diagnosis by screen examination is essential. The ideal position for pictures of the calcification should be estimated on the screen. The projection of choice according to *Roesler* is 15 to 20 degrees of right anterior oblique rotation. *Wosika* and *Sosman* have demonstrated *coronary artery*

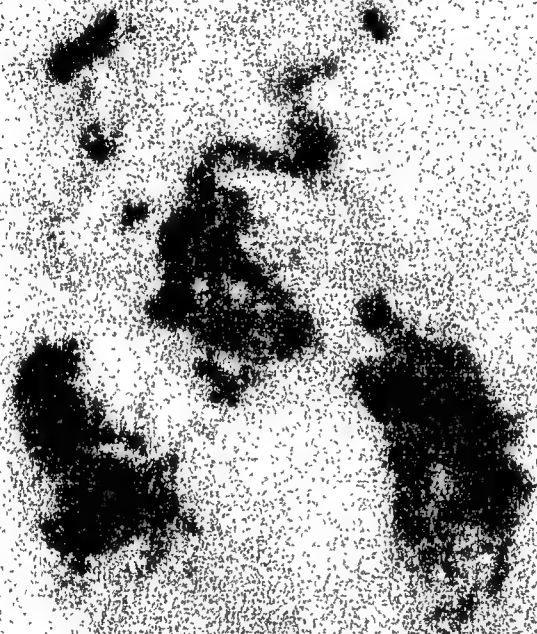


FIG. 33.—Metastatic calcification in the heart. The patient was a young man who died suddenly. No previous history of illness. Aetiology unknown. Case of Dr. Shee.

calcification in three cases *in vivo*. The plaques were linear, segmental, and carved, according to the direction of the vessels. They showed little or no movement and could only be detected on high-speed radiograms. The fact that most individuals with extensive atheroma have calcified costal cartilages makes the demonstration of true coronary plaques very difficult.

Calcification of the *endocardium* is sometimes seen in cases of sub-aortic stenosis and calcification in the *myocardium* sometimes occurs in cases of



FIG. 34.—Calcification of the left auricle in a case of mitral stenosis (first oblique view). The calcification was first observed by Dr Evan Bedford during a screen examination

generalised disturbance of calcium metabolism and rarely in cases of chronic sepsis. Calcification of the *left auricle* is very rare ; Fig. 34 appears to be the only radiographic record of such calcification.

CHAPTER VI

CONGENITAL DISEASES OF THE HEART

IT HAS been estimated by *Schall* and *Diellen* that less than 50 per cent. of cases of congenital heart disease can be diagnosed radiologically. This is due partly to the difficulties in the way of successful X-ray examination of young infants, and partly because many congenital lesions cause no alteration in the radiological size or shape of the heart. A third obstacle to X-ray diagnosis is the frequency with which acquired heart lesions are superimposed on congenital lesions.

For practical purposes congenital heart lesions can be conveniently described under two headings—viz. lesions in which there is no mixture of the arterial and venous streams, and lesions in which there is a free mixture of the arterial and venous streams.

LESIONS WITH NO MIXTURE OF ARTERIAL AND VENOUS STREAMS

In this group we find characteristic radiological appearances in three lesions: (1) Dextrocardia. (2) Co-arcuation of the aorta. (3) Transposition of the aorta. Idiopathic heart hypertrophy has been removed from this group, as the evidence for a congenital aetiology is unsatisfactory.

Dextrocardia

Complete transposition of viscera is rare. The heart is normal in size and shape, and we see a mirror image of the normal. *Kartagener* has published a number of cases of associated bronchiectasis and transposition of viscera, and in one case followed by the author to autopsy there was a well-marked bronchiectasis present. The number of *Kartagener's* cases precludes the possibility of coincidence, and it is likely that the associated bronchiectasis should eventually lead to some dilatation of the conus pulmonalis in the transposed heart. Dextrocardia without transposition of the other viscera is also rare, and in these cases there are usually other congenital lesions of the heart present. *Roesler* describes a true isolated dextrocardia with a mirror image of the heart. He believes that, despite the apparently normal contours of the transposed heart, other defects must be present. In two cases observed by the author the heart was transposed, but not the aorta. One of these patients died, and at autopsy a gross defect of the interventricular and interauricular septa was found. These latter defects were suspected clinically, but the X-ray examination revealed no evidence of them: a surprising fact, as there is usually a gross enlargement of the pulmonary arteries with an interatrial defect (Fig. 35).



FIG. 35.—Dextrocardia without transposition of other viscera. Autopsy revealed wide interventricular and intraauricular defects in addition to the dextrocardia.



FIG. 36.—Dextrocardia with persistence of the duct of Cuvier. The finger-shaped opacity on the left is the azygos vein. A fine line can be seen running from the top of this to the apex of the lung. This line represents the fissure formed when the vein cut through the top of the lung. Case of Dr. Douglas Boyd.

Co-arc-tation of the Aorta

This is a not infrequent malformation of the aorta which is constricted at or just below the level of the ductus arteriosus. The degree of the stenosis determines the 'X-ray appearances. In severe cases a collateral circulation is established through the internal mammary and intercostal arteries, and the latter vessels, dilated and tortuous from increased pressure, erode the inferior borders of the ribs on both sides. These rib erosions or notches were first described by *Roesler* and are pathognomonic of co-arc-tation. *Roesler's* notches must not be confused with the normal arterial furrows on the last inch of the posterior borders of the ribs. In severe cases of stenosis, the actual stricture of the aorta can be visualised on good oblique views. As the stricture is usually just about the bifurcation of the trachea, this bifurcation can be seen with striking clarity.

The left ventricle has increased work to do to force the blood through the narrow vessels making the collateral circulation, and consequently hypertrophies. In many cases, however, there is no evidence at all of enlargement of the left ventricle, even when there is abundant evidence that the stricture of the aorta is very narrow (Figs. 37 and 39).

The ascending aorta is usually dilated and projects well to the right of the middle line. This dilatation of the ascending aorta may assume aneurysmal dimensions, and rupture of the ascending aorta is a not uncommon cause of death in these cases. Curiously enough, the shadow of the aortic knuckle is usually absent or very small. This is probably accounted for by a dilatation of the innominate and subclavian arteries. In two cases seen by the author there was a well-marked indentation on the oesophagus just above the level of the normal aortic indentation. At autopsy this indentation corresponded with a dilated left subclavian artery. In all cases of co-arc-tation of the aorta there is a defect of the middle coat of the arteries, and this is partly responsible for the formation of multiple aneurysms. Such aneurysms in the innominate and subclavian arteries can simulate tumours of the superior mediastinum.

Transposition of the Aorta

This condition seldom occurs as an isolated defect, but is fairly frequently seen in association with other defects. *Bedford* and *Parkinson* have recently published an exhaustive paper on this subject. Eleven cases have come under their notice. There are two types of this defect, one rare and one common. In the commoner type the ascending aorta is more or less central in position, and runs to the right of the trachea and over the right main bronchus. The descending part comes down immediately behind the ascending part, passes behind the trachea, and then lower down crosses over to the right side to pass normally through the diaphragm. There may be a fairly obvious knuckle on the right side and there may be a visible rudimentary left aortic root at the site

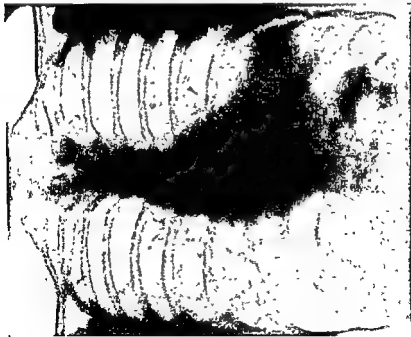


Fig 37. Co aetion of the aorta with gross enlargement of the left ventricle. Death two years later from endocarditis. Autopsy control.



Fig. 38.—Co aetion of the aorta with general enlargement of the heart and congestive failure. Effusion at the left base. Note the notched ribs, especially the left eighth. Death from pre-stenotic rupture of the aorta. Autopsy control.

of the normal knuckle on the left side. This primitive aortic root lies behind the œsophagus and displaces it forwards. In cases of transposition of the aorta associated with other congenital defects, a dilated pulmonary artery usually gives an obvious clue to the nature of the abnormal aortic shadow, but in uncomplicated cases a false diagnosis of mediastinal tumour is usually made.



FIG. 39.—Co-arcuation of the aorta. Atheroma of the descending aorta below the stenosis.

In the postero-anterior view the most striking appearance is not the broad upper mediastinal shadow but the length of the aortic shadow on the right side.

The mistake usually made is to identify this shadow as the ascending aorta, but if it is closely studied it will be seen that it can be followed into the middle line to a point well below the site of the aortic valves. In the oblique views, of course, the diagnosis is obvious, for the whole abnormal course of the aorta can be followed, and a barium swallow shows the œsophagus to be displaced anteriorly. If the aorta becomes dilated or arteriosclerotic, the patient may

suffer from dysphagia. This anterior displacement of the œsophagus is almost pathognomonic of an aortic lesion, and the author can find no record of a mediastinal tumour displacing the œsophagus in this fashion.

The author has seen five cases of anterior displacement of the œsophagus. Two of these were cases of transposition of the aorta, one was an aneurysm, one was a case of spontaneous pneumothorax with rupture of the mediastinum, and one a case of mediastinal abscess complicating puerperal sepsis. In the rare type of aortic transposition there is no persistence of the left aortic root and the course of the œsophagus appears normal. The right-sided course of the aorta can, however, be traced in the oblique views.

FETAL ENDOCARDITIS is a very rare congenital condition. Most workers are sceptical as to the possibility of endocarditis occurring *in utero*, but there is a remarkable specimen in the Royal Chest Hospital which can hardly be explained otherwise. The specimen is the heart of a child aged 2 years, with such a severe degree of mitral stenosis that a probe can hardly be passed through the stenosed orifice. There are no malformations of the valves present. This child was examined radiologically, but there was no X-ray evidence of alteration in the size or shape of the cardio-vascular shadow.

Bicuspid AORTIC VALVE is a comparatively common congenital malformation, but there is no method of determining the presence of this defect *in vivo*.

LESIONS WITH FREE MIXTURE OF ARTERIAL AND VENOUS STREAMS

These lesions are common, and many of them show radiological abnormalities of the cardio-vascular shadow. It must be emphasised, however, that there may be a serious congenital defect present without any radiological evidence of abnormality of the heart.

Patent Interventricular Septum (*Syn. Maladie de Roger*)

This defect is comparatively common, both as an isolated lesion and in association with other congenital defects. A small interventricular defect is of no clinical significance and causes no alteration in the X-ray appearances of the heart. A wide interventricular defect usually results in an arterio-venous shunt, and as a sequel to this the right ventricle enlarges and the pulmonary artery dilates. The aortic shadow is not altered, but the shadow of the superior vena cava may be enlarged and prominent. The main branches of the pulmonary artery may be somewhat dilated. In a general way the heart shadow resembles that of mitral stenosis, but the absence of enlargement of the left auricle eliminates this. Deneke described synchronous pulsation of both borders of the heart as a sign of a wide interventricular defect, but the value of this sign is doubtful, as we are now aware that ventricular pulsation is readily transmitted to the right auricle.

Patent Interauricular Defect

This is an uncommon defect, but causes characteristic X-ray appearances. There is gross enlargement of the right ventricle which swings over to the left side, the aorta is very small, and the conus and pulmonary artery are dilated. Both main branches of the pulmonary artery are dilated, and on the right side this dilatation may reach aneurysmal dimensions (Fig. 41). The smaller pulmonary vessels may be sclerotic and show up clearly, but the loss of lung translucency associated with true passive congestion is absent. Oblique views may show some enlargement of the left auricle. The appearance is not unlike that of aneurysmal dilatation of the left auricle, but in the latter condition all the pulmonary vessels are engorged, with loss of translucency of the lungs, and the two main branches of the pulmonary artery never attain the enormous dimensions seen with an interatrial defect. Not infrequently a pericardial effusion complicates or terminates an interatrial defect, but even a very large effusion does not obscure or account for the gross enlargement of the main pulmonary arteries (Fig. 40). These enlarged vessels are occasionally diagnosed as enlarged glands, and the condition has often been mistaken for Ayerza's disease, a much rarer condition.

Patent Ductus Arteriosus

This malformation is very commonly associated with other congenital lesions. It is characterised by a dilated pulmonary artery which decreases in size with forced inspiration and increases in size with forced expiration. *Roesler* points out that a very large patent ductus may partially obscure the normal aortic window. With a small ductus the heart is not enlarged: with a large ductus the heart is moderately enlarged to right and to left. Calcification often occurs in the aorta and pulmonary artery where the ductus enters.

Pulmonary Stenosis

This, like patent ductus, is rarely seen as an isolated defect. With a severe degree of stenosis the right ventricle is hypertrophied and pushes to the left where it forms part of the left lower pole, giving it a curious pointed appearance. There may be a very deep indentation at the level of the pulmonary artery, and *Assman* shows by an ingenious series of measurements that the main branches of the pulmonary artery are very small, i.e. the hilar shadows appear minute. In other cases the main pulmonary trunk may be normal or grossly dilated and the intrapulmonary vessels may also be normal.

Fallot's Tetralogy

This is a common and well-known congenital lesion characterised by severe cyanosis. If the subject of this malformation reaches puberty or an adult



FIG. 40—Congenital interatrial defect with pericardial effusion. Note the dilation of the main branches of the pulmonary artery and the absence of hyperemia at the periphery and bases of the lungs. Diagnosis confirmed by autopsy.



FIG. 41—Gross dilatation of the pulmonary artery and aneurysmal dilatation of its right main branch. Little hyperemia distal to the main branches. Probably an interatrial defect.

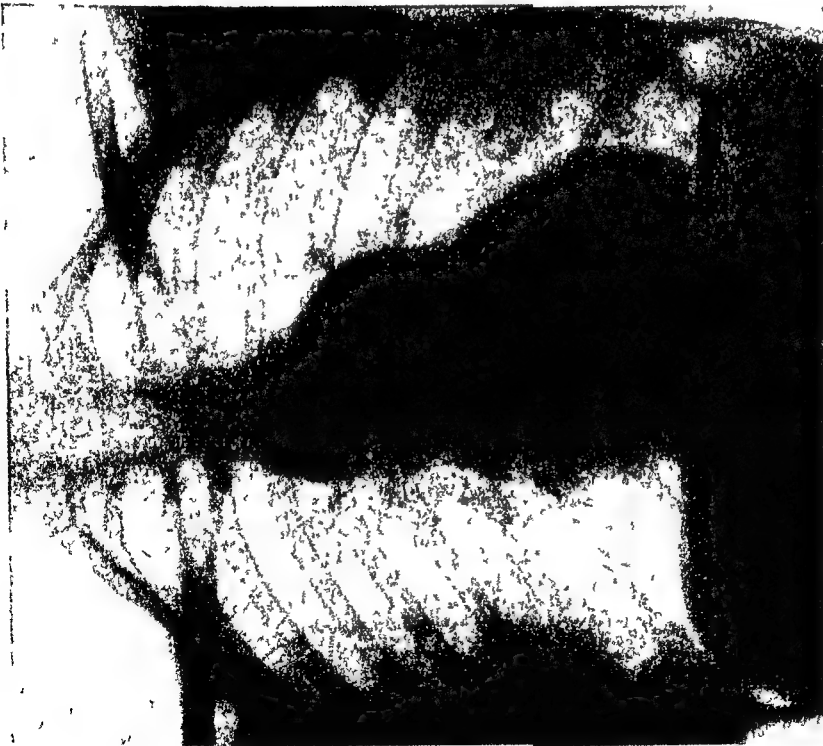


FIG. 42.—Congenital heart disease with gross dilatation of the pulmonary artery and enlargement of the right ventricle, which has swung over to the left.



FIG. 43.—Case of Fallot's tetralogy : note the aortic configuration of the heart and the transposed aorta.

age there are characteristic X-ray appearances. In young children Fallot's tetralogy can seldom be diagnosed by radiology. The tetralogy consists of

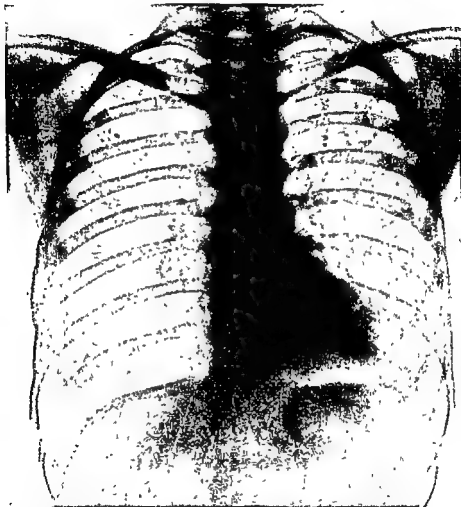


FIG 41 Fallot's tetralogy in a girl aged 16. Note aortic configuration of the heart. Cavity in the left upper lobe and chronic miliary tuberculosis.

pulmonary stenosis, ventricular septum defect, dextro-position of the aorta, and hypertrophy of the right ventricle.

The pulmonary stenosis causes a deep indentation in the heart shadow at the level of the pulmonary artery, the ventricular septum defect plus the pulmonary stenosis cause hypertrophy of the right ventricle. This ventricle pushes the left ventricle to the left and upwards and may actually form a large

part of the left border of the heart. When it does this the apex of the heart can be seen raised well above the diaphragm. The aorta rides over the ventricular defect and has the characteristic appearances of dextro-position, already described. The combination of these defects gives the heart a boot-shaped appearance—the *cœur en sabot* (Fig. 44). This shape is remarkably like the aortic configuration, and indeed an aortic configuration in a young subject should promptly excite suspicion of Fallot's tetralogy. Unlike the true aortic heart,

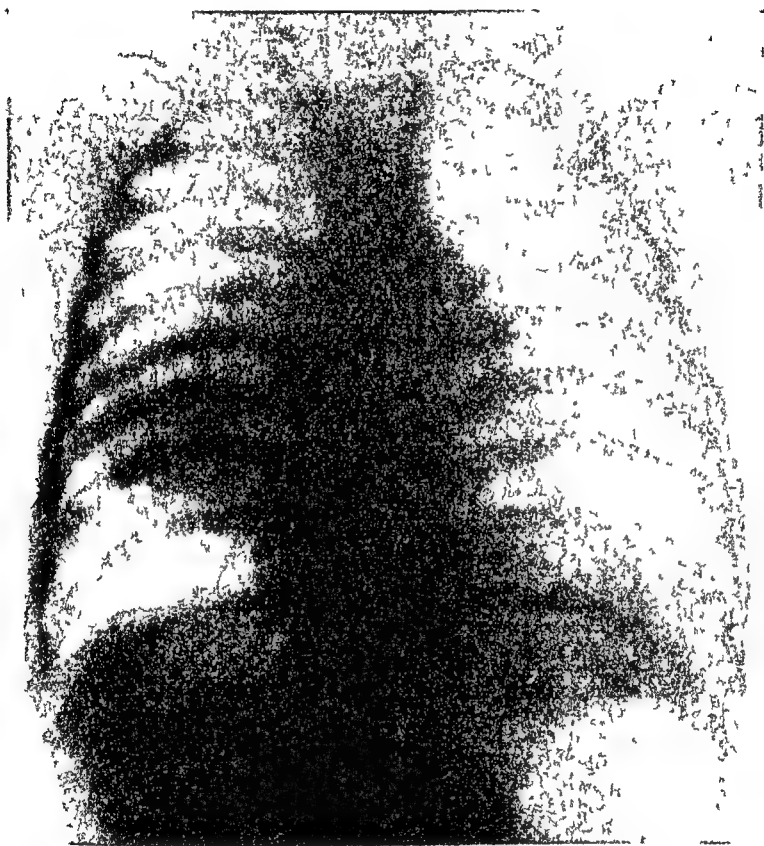


FIG 45 —Cor triloculare.

however, the aortic knuckle is not unduly prominent and the apex of the heart is raised much higher above the diaphragm than ever occurs with aortic disease. Oblique views clearly show that the chamber most affected is the right ventricle. There is some evidence for believing that pulmonary stenosis with or without other defects predisposes to the development of pulmonary tuberculosis.

There are many other gross congenital defects, such as transposition of the arterial trunks and complete absence of the interventricular septum, which

have not come within the scope of radiology, because the subjects of these defects seldom live more than a few days. In some of these defects, however, life may be prolonged for months or even years, and the author has published a case of *cor trilobulare* with characteristic X-ray appearances. The heart was considerably enlarged both to left and to right, there was obvious synchronous pulsation on both borders, and the heart gave the impression of having an apex on each side. The aorta and pulmonary artery could not be identified

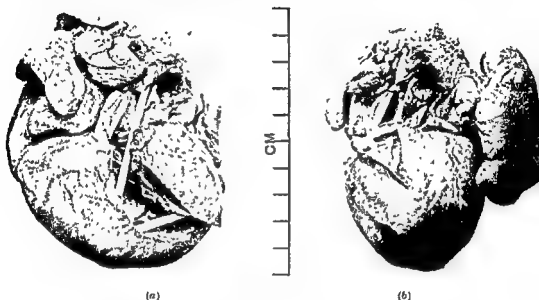


FIG 46 —*Cor trilobulare* (a) shows the single right ventricle and (b) shows the rudimentary left ventricle.

and the auricles appeared normal. These gross defects are of considerable academic interest, but it is the minor anomalies and their effect on the heart which merit most attention. In all cases the condition of the pulmonary vessels should be closely studied. If these are persistently engorged, the prognosis for a long life is unfavourable. Similarly, if the right ventricle is grossly enlarged, the prognosis is not very good. These points are important, as both pulmonary stasis and enlargement of the right ventricle may be present without any clinical evidence of disease. In most patients with a serious congenital defect there is a definite tendency to acquired endocarditis.

CHAPTER VII

THE AORTA

THE NORMAL APPEARANCE

IT HAS already been mentioned that, in the *postero-anterior view*, the normal shadow of the ascending part of the arch of the aorta does not project as a convex opacity on the right side. The ascending aorta and superior vena cava emerge from the pericardial sac together. The vena cava lies on an average half an inch more to the right than the aorta and its inner border is superimposed on the aorta. Thus the base of the vascular shadow on the right side is a combination shadow of vena cava and aorta. An inch to an inch and a half above the outlet from the pericardium the ascending aorta curves inwards over the spine and is no longer visible in the postero-anterior view. The vena cava; on the other hand, swings slightly outwards and becomes more visible as it nears the upper thoracic aperture. There are rare normal individuals with narrow vertebræ and relatively large vessels in whom the whole course of the ascending aorta is clearly visible. In estimating abnormalities of the ascending aorta most workers make use of the following method: Two parallel lines are drawn on the right side, one along the vascular shadow and one along the shadow of the right auricle. The vascular line normally lies well inside the auricular line. If the vascular line is as wide as or wider than the auricular line the ascending aorta is diseased. This rough and ready device is at times useful, but there are many fallacies in its application, notably in cases where the ascending aorta is displaced outwards by tumours, or where tumours overlap and simulate the outline of the vessel.

On the left side the prominent aortic knuckle is situated normally in the second interspace in men and a little higher in women. No great significance can be attached to slight alterations either upwards or downwards, because the aorta is a relatively free organ in the mediastinum; but wide alterations in either direction are pathological. The density of the aortic knuckle is less than the shadow of the left ventricle. This standard of comparison is useful in estimating thickening of the aorta.

The descending aorta examined radiologically is of two types. In the commoner type about an inch of the descending part is clearly visible just below the knuckle—the remainder of the descending aorta is superimposed on the spinal shadow and is invisible. In the less common type about a quarter of an inch of the lumen of the descending aorta projects to the left of the spine and is clearly visible inside the heart shadow as far as the diaphragm. It is seen

as a perfectly straight line parallel to the spine. If it is seen as a convex line, the aorta is senile or pathological. If the pulsation of the normal aorta is studied closely on the screen, it will be noted that at the end of systole on the ventricle the aortic knuckle moves outwards; i.e. the whole of the arch swings to the left during normal pulsation. Fairly forcible pulsation is noted on the knuckle, but, except in the rare cases already mentioned, true aortic pulsation cannot be seen on the right vascular shadow. If the descending aorta is of the visible paravertebral type, no pulsation can be detected on it on the screen, although if accurate radiograms are made in systole and diastole a slight outward deviation can be found in the systolic picture.

In the first or right oblique view the normal aortic arch is fairly clearly visible but in the second or left oblique view the arch is not only equally clear, but its relationship to the trachea and pulmonary artery can be accurately observed. For this reason the second *oblique* view is to be preferred in examining the aorta.

In the second oblique view there are two translucent spaces, known as the aortic window (*Holzknucht*) and the aortic triangle (*Parkinson and Bedford*). (See Fig. 3) The aortic window is a comparatively small space bounded above by the inner concave border of the aortic arch, on the right by the pulmonary artery, and below by the left auricle. An unusually long left main branch of the pulmonary artery may divide this window into an upper and lower compartment. The aortic triangle lies above the arch—its right border is formed by the left subclavian artery, its posterior border by the spine, and its base by the top of the aortic arch. *Parkinson and Bedford*, who first drew attention to this triangle, point out that it is a superior aortic landmark to the aortic window, because it cannot be interfered with by the trachea or left pulmonary artery.

DYNAMIC WIDENING

Dynamic widening of the aorta occurs in young people with rheumatic aortic disease, and also in individuals with hypertension, although in the latter there are usually associated degenerative changes. Dynamic widening is characterised, in the postero-anterior view, by a convex projection of the ascending part on the right side and a high prominent aortic knuckle on the left side. The density of the aortic knuckle is not increased and remains less than the density of the left ventricle, which in both these conditions is often enlarged to the left. Abnormally forcible pulsation is visible both on the knuckle and the ascending part, and the knuckle lies more to the left side than the normal. As widening of the aorta must be accompanied by some shift of the arch as a whole, we find that the aortic window is increased and the aortic triangle is decreased in the second oblique view.

ATHEROSCLEROSIS

Normally with advancing age there is some loss of elasticity of the aorta but it must be emphasised that enlargement of the lumen does not necessarily

follow. When the aorta loses its elasticity it naturally tends to stretch and unfold, so that instead of lying obliquely in the chest it swings round and one is looking so to speak through a true arch. This unfolding of the aorta



FIG. 47.—Kinking of the lower part of the thoracic aorta : note the high unfolded arch.

causes the normal ascending part to project to the right, the aortic knuckle to project higher and more to the left and a prevertebral descending aorta to become paravertebral. It is not accompanied by a

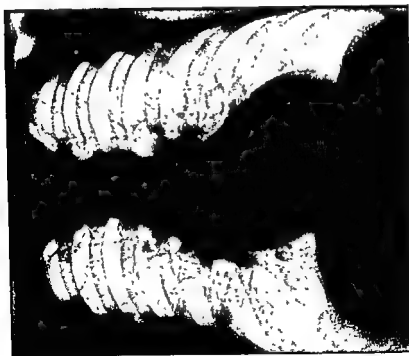


FIG. 48.—Early case of syphilitic aortitis with the ascending part of the aorta irregular in outline and projecting slightly to the right, aneurysm jutting into the right cardio-phrenic angle.



FIG. 49.—Syphilitic aortitis with kinking of the aorta and an aneurysm jutting into the right cardio-phrenic angle.

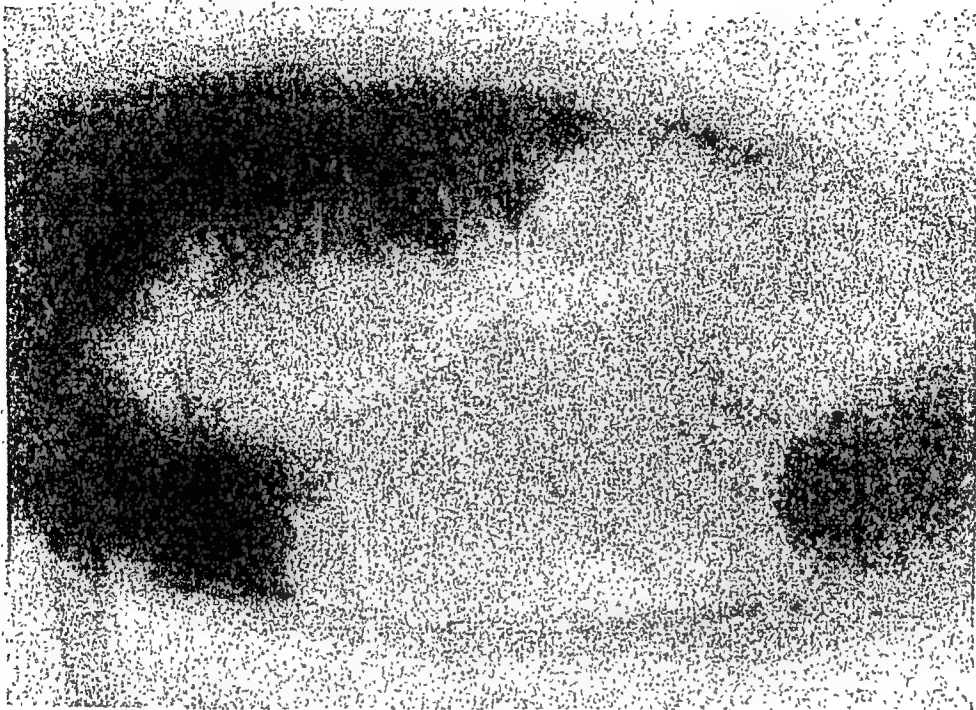


FIG. 51.—Lateral view of a syphilitic aorta showing loss of parallelism.

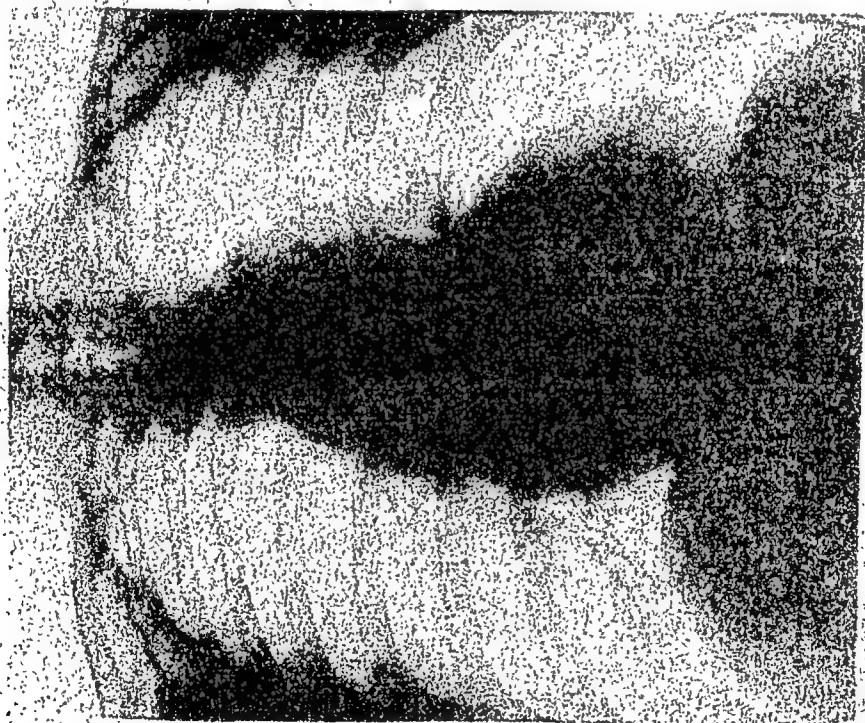


FIG. 50.—Syphilitic aortitis. General dilatation of the aorta and enlargement of the heart. Note the irregular outline of the aorta.

increase in pulsation or density. In atherosclerosis the aorta not only unfolds and elongates, but its density may be considerably increased by calcareous deposits. In the postero-anterior view there is convex enlargement to the right, a high aortic knuckle and a dense paravertebral descending aorta with a convex outer border. In both oblique views the dense arch is very clearly visible. In the first oblique view the thickened ascending part projects well into the retrosternal space. In the second oblique view the aortic window is increased in size and the aortic triangle diminished in size. The arch as a whole takes a wider curve, and the descending part swings well back over the spine. As this descending part must swing back again before it enters the abdomen, it may kink at one point (Fig. 47). This kinking is usually not very prominent in true atherosclerosis, and a marked degree of kinking usually means syphilis. In simple atherosclerosis the aorta is not as a rule dilated, but in some long-standing cases there is slight general dilatation.

SYPHILITIC AORTITIS

The early diagnosis of syphilitic aortitis is of considerable importance, as there is reason for believing that the condition is amenable to antisymphilitic treatment in its early stages. The fundamental lesion in vascular syphilis is replacement of the elastic tissue in the media by fibrous tissue, with compensatory thickening of the intima and adventitia. The condition is patchy, and although the patches may be very extensive the aorta is never uniformly nor universally affected. It is obvious from the pathology that we must seek by radiography to demonstrate localised areas of bulging where the wall is weakened by the syphilitic infiltration. The syphilitic aorta widens, but unless there is associated atherosclerosis it does not unfold and elongate. This absence of elongation is an important diagnostic point.

It is generally agreed that the first radiographic sign of syphilitic aortitis is seen near the root of the aorta. In the postero-anterior view just above the junction of the cardiac and vascular shadows the ascending aorta juts out over the superior vena cava to form a dense convex opacity (Fig. 48). Such an appearance can of course be produced by atherosclerosis with unfolding of the aorta, but the absence of such unfolding can be checked by observing the shape and position of the aortic knuckle on the left side. In the second or left oblique view syphilitic aortitis produces characteristic appearances. In the early cases, with involvement only of the ascending part, this part stands out clearly as a fusiform swelling gradually merging into the normal lumen. Another method of expressing this is to state that the anterior and posterior walls of the aorta do not run parallel to each other. Irrespective of the part of the aorta affected, loss of parallelism of the walls is diagnostic of syphilis. Pure atherosclerosis does not cause loss of parallelism (Fig. 51).

In advanced cases of syphilitic aortitis large areas of the vessel are involved

and it shows general widening. In such cases the shadow of the arch in the postero-anterior view rests like a cap on top of the heart shadow (Fig. 50). As often as not the shadow of the arch is greater than the shadow of the heart itself. If the aortic valves are involved, the left ventricle may, of course, be seen to be considerably enlarged to the left, but it is not uncommon to see severe disease in the aorta with the valves unaffected. Generally speaking, the syphilitic

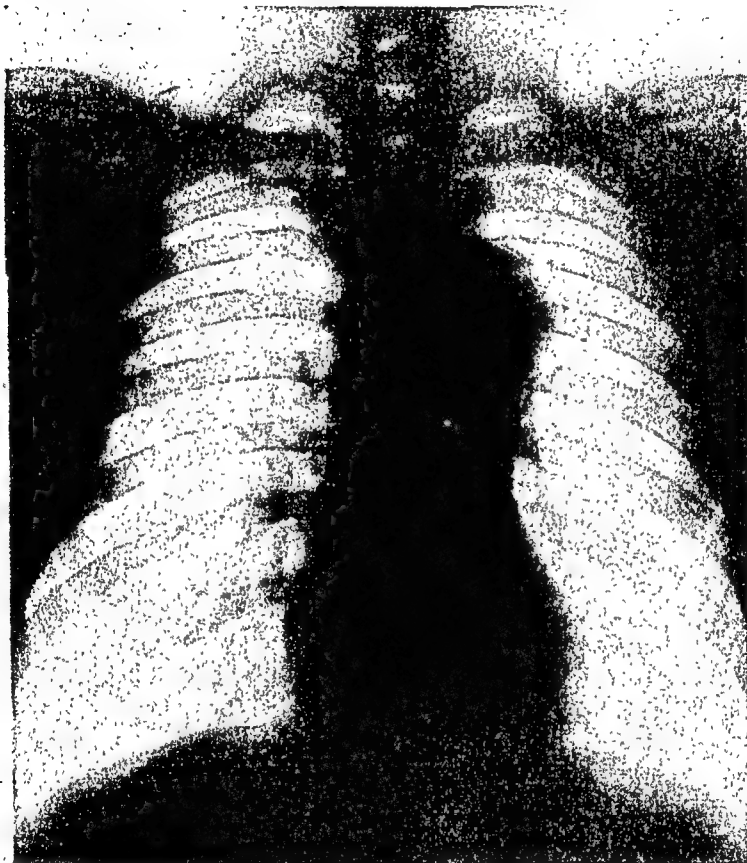


FIG. 52.—Specific aortitis with the descending part of the arch chiefly involved.
Male. Aet. 36. Diagnosis confirmed at autopsy.

aorta is considerably increased in density, but increased density is not a characteristic sign. In long-standing cases there is usually some atherosclerosis with visible calcified plaques and some degree of unfolding and elongation of the aorta. It must be emphasised that syphilitic aortitis produces no abnormal radiological signs in about 30 per cent. of cases.

ANEURYSM OF THE AORTA

Non-syphilitic aneurysms of the aorta occur as a complication of coarctation of the aorta, and consequently they may be seen in young people.

In these rare cases the visibility of rib erosions (Roesler's sign) makes the diagnosis obvious. The vast majority of aortic aneurysms are syphilitic in origin, are seldom seen below the age of 30 years, and usually after the age of 40. They may be large or small, sacculated or fusiform, round or oval, but they are all intimately connected with the lumen of the vessel, and the demonstration of this connection is the vital factor in differential diagnosis. In all cases radiograms should be made in the postero-anterior, antero-posterior, right and left oblique and lateral diameters. The relationship of the normal part and of the abnormal swelling to the œsophagus should also be carefully checked on the screen. The following well-known signs are helpful, but not infallible, aids to diagnosis: A large aneurysm is usually situated along the normal axis of the aorta—i.e. the centre of the tumour usually lies along a line drawn from the aortic knuckle to the junction of the right auricle and ascending aorta. Expansile pulsation is a valuable sign, but as most aneurysms contain organised blood-clot true expansile pulsation is not often seen. Most aneurysms are sharply defined, but in some cases a fairly rapid increase in size of an aneurysm provokes a pleural reaction and the outline, because of pleural adhesions, becomes rather ragged. In most cases of aneurysm the ascending aorta shows some degree of widening, but again this is not an infallible sign, and the author has observed several cases of large aneurysms with the ascending aorta normal in size and outline (Fig. 52). Many aneurysms show sickle-shaped calcified plaques in their walls. Very few aneurysms erode the thoracic vertebræ. When erosion does occur, it is seen as concave defects in the anterior borders of the affected bodies. The discs escape because the condition is not a true destructive one, i.e. osteoclasts remove bone as a response to the constant irritation of pulsation and there are no osteoclasts in the intervertebral discs.

Aneurysms of the Root of the Aorta

Small aneurysms of the sinuses of Valsalva cannot be detected radiologically unless they are densely calcified, and this is very unusual. A large aneurysm of the root of the aorta may project to the left side, and bulge out on the left border just below the aortic knuckle and in front of the pulmonary artery. It pushes the bifurcation of the trachea backwards, but does not affect the œsophagus. It may project so far forwards that it erodes the third, fourth, and fifth costal cartilages. The differential diagnosis from an aneurysm of the pulmonary artery is almost impossible, but the appearance of the smaller pulmonary arteries may help. These vessels are usually unaltered in aortic aneurysms and are dilated with pulmonary artery aneurysms.

Aneurysms of the Ascending Aorta

These are not very common and are usually large. An aneurysm of the ascending aorta appears as a dense shadow with a convex outer border project-

ing into the right lung field. It displaces the aortic knuckle upwards and to the left, and the heart downwards and to the left. As a result of this downward displacement of the heart the lower margin of the aneurysm may apparently lie well below the normal level of the aortic valves, and consequently gives the impression that the tumour cannot possibly have arisen from the aorta. This false impression may lead to erroneous diagnosis of dermoid cyst. Aneurysms of the ascending aorta usually displace the œsophagus and trachea slightly to the right side, but they do not compress these organs.

Aneurysms of the Arch of the Aorta

Small saccular aneurysms springing from the upper or lower borders of the centre of the arch are usually completely invisible in the postero-anterior view. If sufficiently dense and containing calcium they can be visualised in the left oblique view, an aneurysm of the upper border obliterating the aortic triangle and an aneurysm of the lower border narrowing the aortic window.

Large aneurysms of the arch often give a characteristic peg-shaped appearance in the postero-anterior view, and in the left oblique view such aneurysms narrow both the aortic window and the aortic triangle. In some cases large aneurysms of the centre of the arch are accompanied by small pouchings on the ascending and descending parts. These are the most difficult aneurysms to diagnose correctly, as the small pouchings produce a scalloped outline of the upper mediastinum remarkably like the outline of a mass of enlarged glands. Perhaps the most useful method of differentiating between a large aneurysm and a glandular tumour is the position of the œsophagus. Almost all aneurysms displace the œsophagus and trachea; such displacement rarely occurs with lymphadenomatous or lymphosarcomatous tumours. A test dose of X-rays to the mediastinum is also useful. Lymphadenomatous and lymphosarcomatous tumours usually diminish rapidly in size under the influence of X-radiation.

A substernal thyroid is occasionally mistaken for an aneurysm of the arch, but this error can be avoided by noting the level of the aortic knuckle. In aneurysm the aortic knuckle is raised: in substernal thyroid it is depressed.

Aneurysms of the Descending Thoracic Aorta

These are usually easy to diagnose, as in such cases the whole of the vessel becomes paravertebral and the relationship of the tumour to the lumen is visible in the postero-anterior view. "Hard" pictures are necessary in these cases. Most aneurysms of the descending thoracic aorta are fusiform in type and appear as oval opacities through the normal heart shadow. In rare cases the aorta undergoes a sharp kink, and the aneurysm may then project on the right side and be visible through or outside the shadow of the right auricle (Fig. 49). Small saccular aneurysms of the descending thoracic aorta are very unusual.



FIG 53 —Specific aortitis with early aneurysmal dilatation of the ascending part.



FIG. 54 —Aneurysm of the descending part of the aortic arch.



FIG. 56.—First oblique view of Fig. 55. Note the sharp backward kink in the lower few inches of the thoracic aorta.



FIG. 55.—Aneurysmal dilatation of the whole arch of the aorta probably dissecting aneurysm present.



FIG. 57—Specific aortitis with small sacular aneurysm of the descending part. This aneurysm showed no pulsation on the screen. Although the primary aortic lesion was obvious both clinically and radiologically it was thought that the round tumour might be a neurofibroma or a ganglionoma. At operation the round tumour was an obvious aneurysm with forcible expansile pulsation.



FIG. 58 Lateral view of Fig. 57.

Dissecting Aneurysms of the Aorta

These are practically impossible to diagnose radiologically. In rare instances one sees a row of calcified plaques inside a thin bulging aortic shadow.

Aneurysms of the Abdominal Aorta

These aneurysms are often recognised radiologically by the presence of lime salts in their walls. The calcium deposits in abdominal aortic aneurysms may be straight or slightly curved, and may lie longitudinally or transversely to the spine. Granular calcification such as is seen in glands does not occur in these aneurysms. Large aneurysms usually cause erosion of the 12th dorsal and 1st lumbar vertebræ. The differential diagnosis from abdominal cysts is generally easy. The thoracic aorta invariably shows X-ray evidence of disease when there is an aneurysm of the abdominal aorta.

CHAPTER VIII

THE PERICARDIUM

THE NORMAL pericardium casts no shadow, but in certain conditions the shape and size of the heart are so altered that one can infer the presence of pericardial adhesions or fluid in the pericardial sac.

PERICARDIAL ADHESIONS

Extrapericardial Adhesions between the outer wall of the pericardium and the mediastinal pleura are very frequent and occur in many inflammatory diseases of the lungs, and also in association with benign and malignant tumours of the lungs and mediastinum. These extrapericardial adhesions are of little or no clinical or radiological significance. They may cause displacement of the heart, but they do not interfere with its function or cause it to enlarge. Occasionally one sees tag-like projections from the cardiac outline; these tags may represent pleuropericardial adhesions or areas of pleural thickening behind or in front of the heart (Fig. 65).

Intrapericardial Adhesions are often found at autopsy in many types of heart disease, but unless intrapericardial adhesions are gross, they cannot be diagnosed by X-rays. Severe intra- and extrapericardial adhesions occur in the disease known as *chronic adhesive mediastinitis* or *Pick's disease*. In this condition the pericardium is considerably thickened and may be calcified. There are usually thick intrapericardial adhesions and also fibrous tissue formation in the mediastinum outside the pericardium. The condition is sometimes accompanied by polyserositis, and in two of three cases observed by the author there was evidence of a right pleural effusion. The symptoms depend on the degree of constriction of the heart and great vessels. In some cases there is progressive heart failure without evidence of enlargement and without murmurs; in others there are signs of constriction of the superior and inferior vena cava, with enlargement of the liver, ascites (no enlargement of the spleen), and swelling of the neck and face on bending down. Occasionally only one of the two great veins may be constricted.

In a severe case the heart has a characteristic pear-shape. Both borders are smooth and sharp in outline and the normal indentations are missing. The smooth borders on either side run evenly into the aortic shadow, which appears to be much shorter than normal. Engorgement of the superior vena cava may be clearly visible if this vessel is constricted. The transverse diameter of the heart is not necessarily enlarged, and pulsation on both borders is

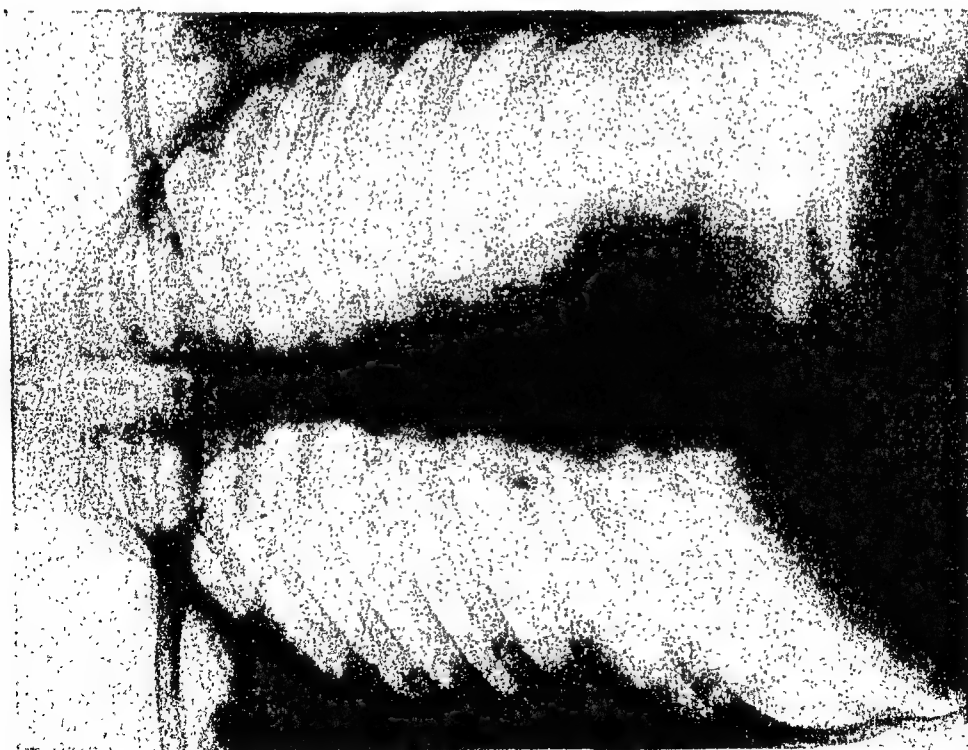


FIG. 59.—External pleuro-pericardial adhesion in the 5th left inter-space anteriorly. History of left-sided pneumonia and pleurisy followed by attacks of paroxysmal tachycardia.



FIG. 60.—Chronic adhesive mediastinitis with irregular calcification of the pericardium. Note the pleural thickening on the right side.



FIG 61—Chronic adhesive mesothorax with calcification of the pericardium. Note the pleural thickening on the right side.



FIG. 62—Lateral view of Fig. 61 showing the thin layer of calcified pericardium encircling the heart.

feeble or completely absent. The pulmonary vessels are not engorged, a striking feature when there is gross enlargement of the liver and ascites. These are the appearances in a severe case, but there are minor degrees in which there is considerable embarrassment of the circulation without any characteristic alteration in the shape of the heart or in its pulsations. It is sometimes possible to determine the existence of such adhesions by demonstrating restriction of the movement of the heart as a whole in different positions and in different stages of respiration. The patient should be examined erect and supine, with lateral and postero-anterior radiograms in both positions. In each position two pictures should be taken, one in inspiration and one in expiration. Complete immobility of the heart during the phases of respiration suggests adhesions, the site of the adhesions being determined by the relative position of the heart to the diaphragm and the sternum in the various views. With anterior adhesions the heart rises with the sternum during inspiration and with diaphragmatic adhesions the heart becomes longer and narrower with obliteration of the normal indentations on deep inspiration. Retrocardiac adhesions usually give no radiological signs. Generally speaking, these signs are not very reliable, but it is likely that X-ray cinematography and kymography will make the diagnosis of pericardial adhesions much easier.

PERICARDIAL CALCIFICATION

In a small percentage of cases of Pick's disease calcification takes place in the thickened pericardium. The calcium is usually laid down as thick plaques, and these may fuse to form a dense band around the whole of the heart shadow. In some cases the calcification may be limited to one or other side; the general view is that calcification tends to be more extensive on the right side, but this was not so in three cases seen by the author, nor does a study of the radiograms of pericardial calcification in the textbooks substantiate this idea. The calcification can be seen in the postero-anterior view, but is best seen in the lateral views. It has to be differentiated from calcification of the left auricle and calcification of a cardiac aneurysm. Calcification of the left auricle is, of course, limited to the auricle, and consequently best seen in the first oblique view. The plaques are easy to localise to the auricle, and in no position can they be superimposed on the anterior borders of the heart where one sees pericardial calcification. Calcification of a cardiac aneurysm is seen around a small bulge just above the apex of the heart. The previous history of a coronary thrombosis in such a case is an obvious guide to the correct diagnosis.

PERICARDITIS WITH EFFUSION

Pericarditis with effusion is common. It has been estimated that between 300 and 400 c.c. of fluid must accumulate in the sac before it can be detected radiologically. It is held by those who do not believe in acute dilatation of the



FIG. 63.—Pericarditis with effusion.

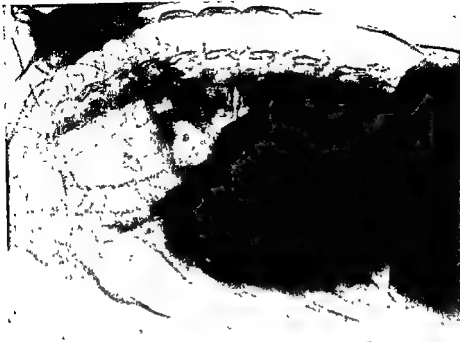


FIG. 64.—Pericarditis with effusion. Second oblique view showing bulging and maximum density in the posterior inferior recess.

heart that erroneous diagnoses of acute dilatation have been made as a result of rapid accumulation of fluid in the pericardial sac. It is doubtful, however, if 300 or 400 c.c. of fluid could accumulate in twenty-four hours without causing death. This problem is complicated by the fact that pericardial effusions frequently complicate enlargement of the heart, and we know that transudates into the pericardial sac can be absorbed over a period of a few days. Most pericardial effusions, however, take weeks or even months to be absorbed if they do not end fatally.

A large pericardial effusion causes considerable enlargement of the transverse diameter of the heart shadow, so that the transverse diameter is greater than the long diameter. It obliterates the normal contours and results in shortening of the vascular shadow, so that we see a very small aorta resting on top of a very large heart. Depending on the quantity of the fluid and the condition of the heart, pulsation is reduced or completely invisible. There may be no stasis in the pulmonary vessels, a useful point in the differential diagnosis from gross enlargement of the heart. *Roesler* describes a very useful sign of effusion, namely distension of the posterior inferior recess. It is obvious that in the erect position fluid will accumulate at the base of the pericardial sac. In the right lateral view the posterior inferior recess of the heart is usually seen as a straight border with the concave inferior vena cava running into it. A pericardial effusion causes a convex bulging of this area. The old idea that the cardio-phrenic angle was obliterated by a pericardial effusion has been shown by radiology to be erroneous.

There are a few reports of localised pericardial effusions on the right side. The condition is very rare and its ætiology unknown. It may be a variety of Pick's disease. A localised pericardial effusion causes a tumour swelling arising usually from the right cardiac border. The differential diagnosis from a mediastinal dermoid cyst is impossible. *Dickson Wright* has described an interesting traumatic cyst of the pericardium. The cyst, which had a calcified border, was about the size of a tangerine and superimposed on the pulmonary artery and conus. It pulsated synchronously with heart and artificial pneumothorax showed it to be extrapulmonary. The cyst was successfully removed and proved to be a blood cyst between the layers of the parietal pericardium. There was a history of a severe blow from a hockey ball over the præcordium.

PNEUMOPERICARDIUM AND HYDROPERICARDIUM

These are usually seen as sequels to paracentesis or mediastinitis. In the latter cases the primary cause of the mediastinitis is an inhaled foreign body or a septic growth of the œsophagus. The radiological diagnosis is simple. The gas-distended pericardial leaves are clearly visible on either side of the central heart shadow. The amplitude of the pulsations of the heart is considerably increased and is transmitted to the fluid level at the base of the pericardial sac.

TUMOURS OF THE PERICARDIUM

There are no radiological descriptions of primary tumours of the pericardium. Mediastinal cysts and tumours are often attached to the pericardium and appear as oval or semicircular opacities projecting from the heart shadow. Malignant infiltration of the pericardium is not infrequent as a complication of carcinoma of the lung or secondary carcinoma of the mediastinal glands. As a rule malignant infiltration of the pericardium is rapidly accompanied by the formation of a blood-stained effusion with the characteristic appearances of an effusion. In a case of carcinoma of the lung observed by the author the heart shadow slowly increased in size over a period of some months. The normal contours of the heart were not obliterated, and at no period was the shape of the heart suggestive of pericardial thickening or pericardial effusion. At autopsy the whole of the pericardium was found to be grossly thickened, and there were less than 20 c.c. of blood-stained fluid in the sac. In another case of bronchial carcinoma, the growth became septic and perforated the pericardium. In 16 days the transverse diameter of the heart increased in size by 4 cms. Pulsation on both borders was normal. At autopsy the heart was found to be normal and there was a thick fibrinous pericarditis with no fluid. This is an instructive case as it shows that normal pulsation may be visible with a severe fibrinous pericarditis.

CHAPTER IX

THE PULMONARY ARTERY

NORMAL APPEARANCES

In the postero-anterior view the normal pulmonary artery lies for its greater part inside the central cardio-vascular shadow, but a small portion may project into the left lung field. This small portion appears as an oval swelling just below the aortic knuckle. *In the first oblique view* the artery can be seen end-on as a dense round opacity just above the root of the aorta. Posterior to this round opacity lies the left main bronchus, which causes a slight normal indentation in the œsophagus. *In the second oblique view* only a small portion of the main pulmonary artery is visible, but its main left branch can be seen running transversely across the aortic window. The right and left main branches of the pulmonary artery are chiefly responsible for the hilar shadows. The bronchi and pulmonary veins play no material part in the production of the hilar shadows.

The faster the speed at which the radiogram is taken, the clearer the definition of the arterial shadows ; but because numerous branches spring off in all directions, the two main vessels are not seen as well-defined tubular shadows. The position of the two main branches varies considerably, and in this connection it must be emphasised that the pulmonary vessels in virtue of their own elasticity and their elastic bed, the lungs, are capable of wide ranges of movement. In the average chest the main arteries jut into the lung fields almost at right angles to the central shadow and then tilt slightly downwards and outwards. In long narrow chests this tilt is slightly exaggerated, and in broad chests the tilt is diminished. The arteries are lower during inspiration and higher during expiration. They are also higher in the prone than in the erect position. Irrespective of their height or angle of inclination in the lungs the left main branch is always about half an inch higher than the right. This relationship is constant except in certain diseases of the lungs, when one or other vessel may be pulled upwards or downwards by fibrous tissue. If one branch is pulled up, its minor branches are seen to run straight downwards instead of obliquely downwards and outwards. If one branch is pulled or pushed down, its minor branches have a more concave direction and are crowded together.

Assman, who has done considerable work on the shape, size, and direction of the main pulmonary arteries, has evolved an accurate system of measurement of their size. His figures have been obtained from radiograms taken in

the erect position at a distance of 1.5 metres. He estimates the width of the average hilum between 11 and 14 mm., and states that anything over 15 mm. and below 10 mm. is pathological.

ENLARGEMENT OF THE PULMONARY ARTERY

Enlargement of the main pulmonary artery may be present without any alteration in its lesser branches, or may be associated with decrease or increase in size in them. The condition of the smaller branches is determined by the nature of the disease causing enlargement of the main trunk. Enlargement of the main pulmonary artery is seen in the postero-anterior view as a large semicircular swelling projecting over the left hilum, immediately below the aorta. *In the first oblique view an enlarged pulmonary artery is seen as a round dense opacity, often pushing the left main bronchus downwards. Its increased size usually renders it greater in density than the heart shadow itself in this position. If a barium swallow is given, it will be observed that the normal indentation of the left main bronchus on the œsophagus is increased. Assman has shown that an enlarged pulmonary artery can compress the left upper lobe vein, and in the postero-anterior view this may be seen as increased vascularity and decreased translucency of that lobe.*

The causes of enlargement of the pulmonary artery are as follows :

(1) **CONGENITAL HEART DISEASE.**—In most cases with a very free admixture of the venous and arterial streams, the main trunk and its lesser branches are enlarged. In cases of pulmonary stenosis, the main trunk alone may be dilated, and the lesser branches may be normal or diminished in size. In cases of coarctation and dextroposition of the aorta, the absence of the aortic knuckle on the left side gives a false impression of increase in size of the pulmonary artery.

(2) **ANEURYSM OF THE PULMONARY ARTERY.**—This is rare and is always of syphilitic origin. A few proven cases are on record, and the following are the radiographic findings. The aneurysm appears as a large semicircular tumour jutting out from the mediastinum just below the aortic knuckle. It hides the left hilum and may be big enough to conceal the aortic knuckle. The tumour may or may not show vigorous pulsation. The vascular markings in the left lung are usually increased in size and density. As the tumour comes forward it may erode the costal cartilages above it. In the first oblique view the aneurysm bulges into the retrosternal space, and posteriorly its pressure on the left main bronchus causes a considerable increase in the depth of the left main bronchus impression on the œsophagus. In true aneurysm of the pulmonary artery there is usually less blood flowing into the right main branch, and consequently the right hilum shadow may measure less than 10 mm. The differential diagnosis is from aneurysm of the root of the aorta, which, as previously mentioned, may project forwards and to the left over the pulmonary artery. Short of injecting some opaque substance into the aneurysm and

observing the direction it flows under the screen, there is no accurate method of differentiating between the two conditions.

(3) RUPTURE OF AN AORTIC ANEURYSM INTO THE PULMONARY ARTERY is most unusual. One case has been observed by *Wimberger*. Gross dilatation of the pulmonary artery followed the leakage.

(4) FISTULA BETWEEN THE PULMONARY ARTERY AND THE AORTA may



FIG. 65.—This is probably a true case of aneurysm of the pulmonary artery. Oblique view showed no displacement of the œsophagus or trachea, and a normal descending aorta could be visualised. The aneurysm eroded three ribs anteriorly and formed a large pulsatile swelling under the skin. Aneurysm of a sinus of Valsalva cannot be excluded for certain. Female. Aet. 36. W.R. + + .

occur as a result of syphilitic ulceration. The condition is mechanically the same as a patent ductus arteriosus. In a case seen in the Royal Chest Hospital, there was gross dilatation of the pulmonary artery, enlargement of the right ventricle, and stasis in the lungs. All the smaller pulmonary vessels showed degenerative changes, and there were large calcified plaques visible in the main trunk. At autopsy it could not be determined whether the condition was syphilitic or congenital in origin.

(3) **MITRAL STENOSIS.**—In this disease there is usually considerable enlargement of the pulmonary artery and engorgement of its smaller branches.

(6) **IN ALL CASES OF CONGESTIVE FAILURE**, irrespective of the primary condition, there is slight enlargement of the pulmonary artery.

(7) **IN CASES OF CARDIAC ENLARGEMENT ASSOCIATED WITH ENDOCRINE OR DEFICIENCY DISEASES** there is usually some enlargement of the pulmonary artery. The enlargement may be considerable, and is not associated with changes in the lesser branches.

(8) **IN ASTHMA, EMPHYSEMA, AND SCLIOSIS** there is often a dynamic swelling of the pulmonary artery.

(9) **IN ALL CHRONIC INFECTIONS OF THE LUNGS** there is some destruction of the vascular bed, and in addition the vessels which escape destruction may develop endarteritis. The combination of the two factors leads to swelling of the pulmonary artery and enlargement of the conus pulmonalis. In carcinoma of the lung a large branch, or even the main trunk, may be constricted by growth with consequent dilatation of the artery.

Calcification of the pulmonary artery is not uncommon when the vessel has been enlarged for a considerable time. Such calcification is seen as sickle-shaped or semicircular plaques equally visible in the postero-anterior and first oblique views.

HYPERÆMIA OF THE LUNGS

So-called congestion of the lungs is a loose clinical term which might well be abandoned, because it embraces many diverse conditions and often leads to confusion and false diagnoses. Hyperæmia of the lungs may be active or passive and may be local or generalised.

Passive Hyperæmia arises when the flow of blood from the lungs to the left ventricle is impeded, the common causes being mitral stenosis and cardiac failure. In the initial stages of the condition only the pulmonary veins and capillaries are engorged, but, as the right ventricle hypertrophies to overcome the resistance in the venous circulation, the pulmonary arteries also distend. When both sets of blood-vessels are involved, transudation occurs into all the lymph-vessels, and finally in severe cases the bronchial arteries are affected, with resultant cedema of the bronchial mucosa.

The X-ray appearances vary enormously, because there are undoubtedly many types and degrees of passive hyperæmia. In the commonest type the main hilum shadows are enlarged and less well defined than normal. Lobar vessels seen end-on may be as big as a pea and so dense that they are often erroneously interpreted as calcified glands. The smaller branches are clearly visible right out to the edge of the chest wall, and as a result of swelling of the smaller vessels coupled with exudation into alveoli there is marked diminution in the translucency of the lungs. It is impossible to get a picture with good contrast. On the screen the diaphragmatic movements are seen to be restricted,

and there is diminished expansion in all parts of the lungs. If the lymphatics are engorged, apparent thickening of the inter-lobar pleura can be visualised in all the fissures. This is due to swelling of the large lymphatics which connect the parietal and visceral pleura through the fissures. Occasionally the combination of swollen end-on vessels and alveoli filled with heart-failure cells causes a miliary appearance in both lungs. This miliary appearance can be remarkably like pneumoconiosis or miliary tuberculosis, but can be differentiated from these conditions by the fact that the spots in the vascular disease

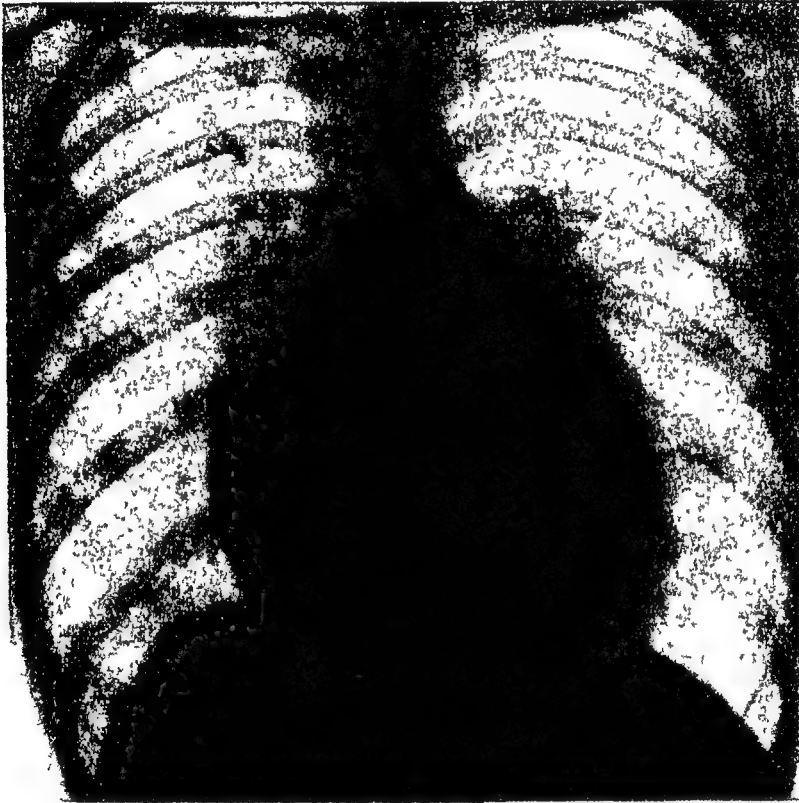


FIG. 66.—Mitral stenosis with the miliary type of passive hyperæmia of the lungs.

decrease in size towards the periphery. In pneumoconiosis and miliary tuberculosis there is never the marked loss of translucency seen in passive hyperæmia of the lungs.

There is a rare type of passive hyperæmia of the lungs seen in some cases of mitral stenosis. Instead of the gross swelling of the hilar shadows and the coarse increased density and arborisation of the smaller vessels to the periphery, one sees a fairly fine mesh appearance radiating from the hila to every corner of the lungs. The striæ are not so wide as vessels and are denser and sharper in outline, and there is no marked decrease in the translucency of

lungs. The appearances are identical in every respect with those seen in lymphangitis carcinomatosa, malignant infiltration of all the lung lymphatics. The common type of passive hyperæmia seen in mitral stenosis seldom disappears completely under treatment, but this "lymphatic" type does disappear com-

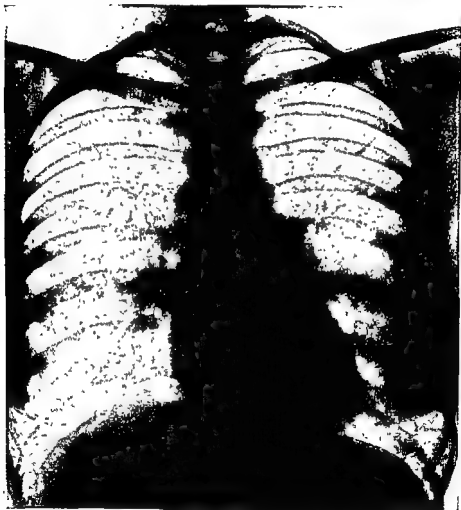


FIG. 67—Mitral stenosis with the "lymphatic" type of passive hyperæmia of the lungs.

pletely. The underlying pathology of this rare form of passive hyperæmia has not been discovered, but it is not improbable that it is a true lymphatic obstruction caused by an enlarged left auricle pressing on the thoracic and right lymphatic ducts.

Localised passive hyperæmia of the lungs occurs in the larger vessels about

the hila in the early stages of congestive heart failure. The swollen vessels are easy to identify on good radiograms. This localised hyperæmia is a sign of considerable importance, as it indicates impending failure long before clinical evidence of failure is suspected. In congestive heart failure all the previously described signs of passive hyperæmia are intensified and in addition fairly

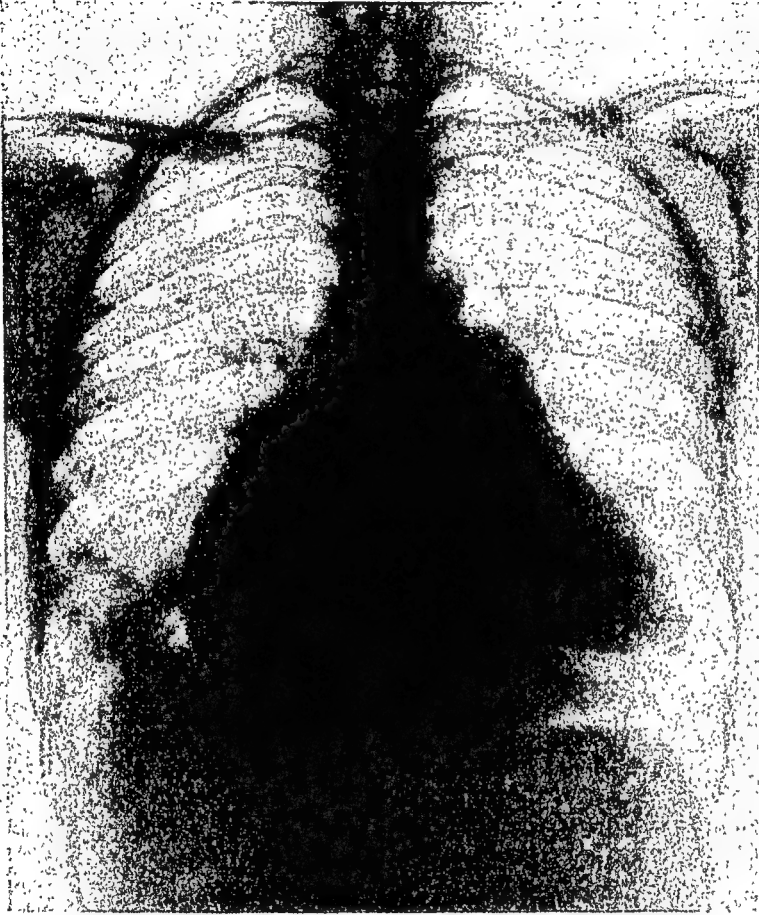


FIG. 68.—Combined mitral and aortic disease with pericardial effusion and resolving infarct at the right base.

large opaque areas are visible in the lungs near the roots. These opacities are small areas of collapse, due partly to oedema of the bronchial mucosa and partly to compression of lobules by distended capillaries. Collapse of lobules occurs near the roots of the lungs because that is the situation of minimum elasticity. Pleural effusions are, of course, a frequent complication of congestive heart failure. Unlike the chronic pulmonary stasis seen in mitral stenosis and congenital heart disease, the stasis in congestive heart failure can disappear completely under treatment.

In cardio-vascular-renal disease one occasionally sees hyperemia localised to the main vessels and accompanied by large areas of loss of translucency, the

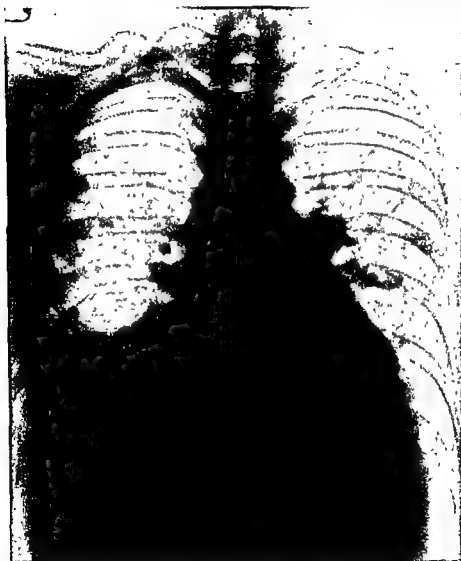


FIG 69—Cardio-renal disease. Sub-acute edema of the lungs with atelectatic area in the right lower lobe

latter being caused by excessive exudation into the alveoli. The peripheral parts of the lungs are normally translucent. This condition can occur very rapidly and also disappear very rapidly under treatment. It is often compl-

cated by hæmoptysis. It is hardly a true passive hyperæmia, and is perhaps best described as subacute pulmonary œdema.

Active Hyperæmia of the Lungs occurs in all inflammatory processes of the lungs and pleura, and in such cases radiological signs of it are usually completely obscured by the primary disease. It also follows inhalation of many poisonous gases and of steam. Although the immediate effect of inspiration of these substances is acute hyperæmia, bronchiolitis or broncho-pneumonia so quickly supervenes that the picture in these cases is that of the secondary lesion.

Mechanical changes in the pressure relations in the alveoli also cause active hyperæmia (*Kaufmann*). In airmen who are flying at a great height and also in mountaineers the rarefaction of the air creates a negative pressure in the alveoli, and consequently an increased pressure in the capillaries, thus causing active hyperæmia which may lead to hæmoptysis. Similarly, if a pleural effusion compressing the lung is drawn off too quickly, the induced negative pressure within the thorax may cause sufficient hyperæmia to lead to hæmoptysis. In circumstances such as these the hyperæmia is only temporary, and subsequent radiological findings are negative.

From the radiologist's point of view the most important form of active hyperæmia is the so-called collateral or fluxionary hyperæmia, which occurs when the circulation in a large area of lung tissue is obstructed. This is seen *par excellence* in the healthy lung in a case of artificial pneumothorax, and also in the healthy lung when the other side is compressed by a large pleural effusion. The X-ray appearances of this form of hyperæmia can be very misleading and closely resemble those of pulmonary tuberculosis. The swollen vessels cause slight general loss of translucency, and those which are projected obliquely and end-on are very like foci of tuberculosis. Indeed, the resemblance may be so close that a differential diagnosis is impossible.

Another form of hyperæmia which is very confusing to the radiologist is that which follows pneumonia and whooping-cough. Although clinical signs may be completely absent, X-ray examination shows that hyperæmia of the lung persists for some months after all subacute and acute lung infections. This manifests itself as loss of translucency, increase in size of the vessels, and diminished movements over the affected area. It can be easily identified in an adult giving a history of a recent pneumonia, but in children, where it is a constant sequel to broncho-pneumonia and bronchitis, it can easily be confused with bronchiectasis. The larger pulmonary vessels are most affected, and these, running side by side and clearly visible through the heart shadow, give an impression of dilated bronchi. Often a lipiodol is necessary to make the differential diagnosis, but the following point is of great help: In post-pneumonic hyperæmia there is rarely compensatory emphysema; in basal bronchiectasis in children there is nearly always compensatory emphysema.

It is obvious that active hyperæmia must be present in some degree in all cases of pulmonary tuberculosis, and there is little doubt that this is often mistaken for recent tuberculous infiltration.

Even the most experienced observers find difficulty in estimating the point at which true infiltration stops and hyperæmia begins. Most often one sees an upper zone the seat of chronic fibroid phthisis with cavitation, and below this and merging with it into the middle and lower zones an area of hyperæmia. If the tuberculous foci are of the so-called productive type, they appear as small comma-shaped nodules easily differentiated from a swollen vessel. The exudative tuberculous focus is usually bigger than an end-on vessel and also less well defined. Another helpful point is the fact that tuberculous foci tend to group themselves together and coalesce.

French authors describe a primary hyperæmia of the lung known as the *maladie de Woulez*. Young and Beaumont believe that this is a mild or abortive pneumonia. In similar cases which have come under my own observation the disease proved to be thrombo-phlebitis migrans.

CHAPTER X

THE PERIPHERAL VESSELS

THE PERIPHERAL vessels may be visualised directly if calcium is deposited in their walls. Such calcification is commonly seen in elderly people and in diabetics. It may also occur in any of the diseases associated with general disturbance of the calcium metabolism. The calcification is usually best seen in the legs and arms, and in the internal iliac arteries. Extensive calcification may be visible without any clinical evidence of interference with the circulation, and indeed in cases of intermittent claudication and thrombo-angeitis obliterans calcification is usually conspicuous by its absence. In rare cases the splenic artery alone is susceptible to degenerative changes. Arteriosclerotic aneurysm of the splenic artery may be symptomless until it ruptures or may be associated with left upper abdominal pain. *Haffner* has collected 86 cases from the literature. Of these, only 11 who were operated on before rupture survived, and of the 11 only 4 were correctly diagnosed before opera-



FIG. 70.—Atheroma of the popliteal artery—no vascular symptoms.

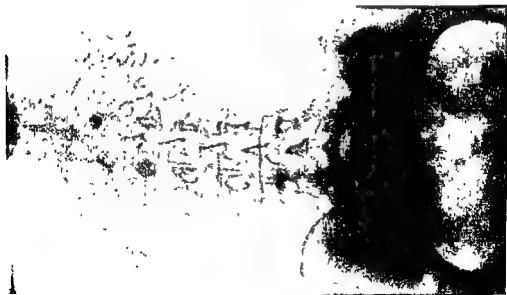


FIG. 71.—Arteriosclerotic aneurysm of the splenic artery. Case of Dr. C. J. ...



FIG 72.—Atheroma of the abdominal aorta in a man suffering from osteitis fibrosa cystica.

tion. In the case illustrated in Fig. 72, the artery is diseased in its full length. Smaller aneurysms of the splenic artery appear as round, oval, or cylindrical calcifications in the region of the hilum of the spleen. The differential diagnosis is from calcified cysts or infarcts in the spleen, calcified glands, pancreatic stones, and aneurysm of the renal artery.

The peripheral vessels can be examined in detail only if a radio-opaque solution is injected into them. This procedure is known as *vasography* or *arteriography*. It has been practised much more on the Continent than in England, and for complete details of the various methods the reader is referred to a recent excellent publication by *Reboul*. The substances commonly used for injection are Thorotrast and the iodine compounds, Uroselectan B., Abrodil, and Tenebryl. Thorotrast is a colloidal solution of thorium dioxide, harmless to the vessels but retained in the liver, the spleen, and the bone marrow. It is a radioactive substance, and has been proven to be a carcinogenic agent in animals. For this reason its use in human subjects is being abandoned. The various iodine compounds are harmless in the vessels, but cause such intense pain on passing through the capillaries that they can only be injected under a general anæsthetic. Arteriography is still in an experimental stage, and there have been several unfortunate sequelæ, such as gangrene of a limb and exacerbation of arteritis. Doubtless the possibility of such accidents will soon be eliminated, and the method will then be of great value both to clinician and surgeon.

The chief value of arteriography is in the localisation of emboli, which appear either as complete block of a vessel or as an oval unilateral narrowing of the lumen. It is also valuable in localising small peripheral aneurysms and in differentiating between spastic and organic vascular disease. In senile and diabetic arteritis arteriography shows the vessels to be exceedingly tortuous and irregular, with filling defects in their walls. They may be so reduced in size that they are threadlike. In thrombo-angitis obliterans the calibre of the larger arteries is progressively diminished, the walls are irregular, but the vessels as a whole are not tortuous and there is a liberal collateral circulation.

Arteriography has also been used in the study of the circulation in malignant tumours of the limbs, and around arthritic joints and areas of osteomyelitis.

BIBLIOGRAPHY

- ASSMAN, H., "Die Klin. Röntgendiagnostik d. Innererkrankungen," 5th edn., Berlin 1934.
 CIGNOLINI, P., "Cardiac Kymography," *Radiol. Med.*, 1932, 177.
 DAVIES, D., HODGSON, G. H., and WHITBY, L., "Pneumonia," *Lancet*, April 1935, 919.
 DEAN, J., and DEAN, J. C., "Arteriovenous Aneurysm," *Wisconsin Med. J.*, Aug. 1934, 587.
 DENEKE, T., See ST. ENGEL'S "Handbook of X-ray Diagnosis and Therapy in Children," Leipzig, 1933.
 DICKSON WRIGHT, A., "Calcified Cyst of the Pericardium," *Brit. J. of Surg.*, 1936, 612.

- DIETLEN, H., and SCHALL, L., ST. ENGEL'S "Handbook of X-ray Diagnosis Therapy in Children," Leipzig, 1933.
- DORNER, "Clinical Studies on the Pathology and Treatment of Diphtheria," Jena, 1
- EHRENBERG, "Two Cases of Tumours in the Heart," *Deutsch. Arch. f. Klin. u. CIII*, 293
- EVANS, W., "The Course of the Oesophagus in Health and in Disease of the Heart Great Vessels," *Med Research Publications*, No. 208, 1937.
- HAFFNER, J., "Calcified Aneurysm of the Splenic Artery," *Acta Radiol*, 1936, 602
- KAUFMANN, E., "Text-book of Pathology for Students and Practitioners," London, 1
- KNOX, R., "The Investigation of the Movements of the Heart by the Use of Slt Diaphr and the Moving Film," *Brit. J. Radiol*, 1925, 142.
- KRAUSE, "Cardiac Tumours," *Atlas. d. Röntgendiagnostik*, Groedel, Munich, 1914
- PALMER, J. H., "The Development of Cardiac Enlargement in Disease of the Hea *Med Research Publications*, No. 222, 1937
- PARKINSON, J., "Enlargement of the Heart," *Lancet*, 1936, 1337 and 1391.
- PARKINSON, J., and BEDFORD, D., "Right sided Aortic Arch," *Brit. J. Radiol.*, 1936, 7
- "The Aortic Triangle," *Lancet*, Oct., 1936.
- PARKINSON, J., and HOYLE, C., "The Heart in Emphysema," *Quart. J. Med*, Jan, 11
- RABINOVITZ, L., and ROGERS, E., "The Heart in Deficiency Diseases," *New Eng Med*, 1936, 621
- REBOUL, H., "Arteriography," Paris, 1935.
- ROESLLER, H., "Clinical Roentgenology of the Cardiovascular System," London, 19
- Heart Section in ROSS GOLDEN'S "Diagnostic Roentgenology," New York .
- Edinburgh, 1936
- RUBIN, E. L., "The Size of the Heart in Asthma and Emphysema," *Lancet*, Nov. ' 1936.
- RUSSELL REYNOLDS, J., "Cmeradiography," *Brit. J. Radiol*, 1934, 415.
- SABAT, B., "The X ray Demonstration of Moving Organs," *Fortschr. a. d. Ges. d. R genstrh*, 1913, 42.
- STUMPF, P., "Rontgen Kymography," *Brit J. Radiol*, 1934, 707
- WENCKEBACH, K., See ASMAN, Heart Section.
- WIMBERGER, K., "Rupture of the Aorta into the Pulmonary Artery." See ASMAN Heart Section
- WOSKA, P., and SOSMAN, M., "The Roentgen Demonstration of the Coronary Arte in Living Subjects," *J. Amer. Med. Assoc*, 1934, 591; "Intracardiac Calcificatio *Amer. J. Roentgenol.*, Jan., 1934.

PART TWO
RESPIRATORY SYSTEM

BY

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PART TWO

RESPIRATORY SYSTEM

CHAPTER XI

RADIOLOGICAL INVESTIGATION OF THE CHEST

THE RADIOLOGICAL investigation of the chest consists of two parts, fluoroscopy and radiography. These are complementary to one another, and neither can safely be omitted. In a few rare cases the patient is so acutely ill that it is only possible to take radiograms as he lies in bed. In this case, if he cannot turn on to the side, a sufficient number of films must be taken without moving him, lateral and oblique films being made with suitable displacement of the tube. In the great majority of cases, including bedside examinations with a portable apparatus, screen examination is practicable and essential.

The special facilities which are provided in an X-ray department render it advisable that bed cases should be brought there for examination. The best procedure is to transport the bed on a special wheeled trolley. The head of the bed should be removable, so that the patient may be examined sitting up at the screening stand, which should be adapted to this purpose. The great majority of bed patients are examined in this way in the writer's hospital practice.

Bedside screen examination in the patient's home may be carried out very efficiently and completely with a portable apparatus if care is exercised and a few simple arrangements are made beforehand. If the examination must be conducted in the daytime, the radiologist's eyes must be "adapted," and he should wear dark-red glasses for ten minutes before starting to screen the case. If the blinds and curtains of the room are drawn, and most of the light excluded by pinning up sheets of brown paper over the windows, quite adequate screening may be carried out with the help of a cryptoscope, which consists of a pyramidal light-tight box attached to a portable fluorescent screen. If this is not available, an excellent substitute is found in an ordinary dark cloth as used by photographers, or even, in case of emergency, a blanket. If the cloth encloses the observer's head and the screen, he finds himself in a light-tight tent, in which it is easy to make a fluoroscopic examination. An experienced observer, who has acquired by long practice under routine conditions the facility of quickly grasping the significance of what he sees on the screen, is then able, by screen examination in many planes, to come to a rapid decision as to the type of lesion present, and to plan his radiographic procedures

accordingly. The serious condition of most of these patients, and the need, in many cases, for early surgical intervention, render it imperative that the examination should be controlled by fluoroscopy and that great pains should be taken with it.

TECHNIQUE OF SCREEN EXAMINATIONS IN AMBULANT CASES

The patient is instructed to remove the clothing down to the waist, and must be provided with a dressing-gown of light non-opaque material. Hospital cases should bring their clinical notes, and any previous films which are available. The radiologist should, if full notes are not available, take a history of the case, and should make such clinical examination as he considers will be of value as a preliminary to the radiological study. He need not be deterred therefrom by any feeling that he is usurping the function of the clinician. It is rather his duty to possess himself of any clues which may help to make his examination more efficient and thorough. If he approaches the case at the outset from the same angle as his clinical colleague, and is aware of the clinical problem, he will be more cautious in his interpretation, less liable to jump to conclusions, and less inclined to be satisfied with the obvious. If there is a discrepancy between the clinical and the radiographic findings, this should increase his sense of responsibility and prompt him to search for the cause of the discrepancy. In essence his task is to elicit and to evaluate the radiological signs. This task he will perform better if he is not oblivious of the other physical signs. If the subsequent integration of clinical and radiological findings is made by personal consultation, the patient, the radiologist, and the clinician all gain much thereby.

Whether the radiologist elects to make a clinical examination or not, he will at least not omit to observe the following points, among others: dyspnoea, cyanosis, clubbing of the fingers, tachycardia, type and rate of respiration, deficient or altered respiratory movement, cough and pain. He should examine the chest for deformities of the bony thorax, scoliosis, kyphosis, cervical ribs, tumours of ribs, breast or other soft parts, and should look for scars, œdema or local swellings, enlarged veins, enlarged glands in the neck, axillæ, and groins, enlargement of the liver and spleen, abdominal tumour, ascites. He should note the size of the female breasts, and whether one or both breasts have been removed. He should assess the general condition of the patient and his fitness for prolonged routine examination. If the patient is not fit, the examination must be modified accordingly.

Barium paste and fluid barium suspension should be at hand, since examination of the mediastinum and of the heart and great vessels is incomplete unless the œsophagus is filled. In diaphragmatic displacements and hernia, œsophagectasia, thoracic stomach, and many other conditions, it is indispensable.

A preliminary screening of the nasal sinuses is a useful procedure. Opacity of the maxillary antra may be thus detected and throw light upon an obscure

respiratory infection. If any abnormality is noted or suspected on clinical grounds, films should be taken of the nasal sinuses.

Under ordinary conditions the examination of the chest is conducted with the patient standing or, if unable to stand, sitting up. In the erect position the diaphragm descends more completely and the basal parts of the lung are better aerated. It is also easier to rotate the patient into the oblique positions so essential to a complete study of the lungs and mediastinum, and fluid levels in cavities or in the pleural sacs are more easily observed. Should the patient be unable to maintain the erect position for a sufficiently long time, arrangements should be made to permit examination in the postero-anterior plane while he lies on the side. In this way fluid levels are readily demonstrated, and valuable additional information is obtained in some special cases of hydro-pneumothorax and subphrenic abscess.

The examination commences with a general survey with fully open diaphragm-shutter while the patient breathes quietly. Note is taken of the general conformation of the chest, the position of the heart and mediastinum, of the leaflets of the diaphragm, and of the diaphragmatic movements. The patient is then instructed to inhale and exhale deeply and the observations are repeated, special attention being given to the "air entry" into different parts of the chest, as judged by the degree of illumination on inspiration. Inspiratory displacement of the mediastinal shadow to one side is looked for. Minor degrees of such movement may easily be overlooked; it is, however, an important sign in bronchostenosis, and in pneumothorax. If one lung field remains bright on expiration, a pneumothorax or a valvular bronchial obstruction may be present. A partial pneumothorax may very readily be missed if it is confined to the surface and the lung is little collapsed. It is more easily seen during full expiration. The observer should also note the marked difference of visibility of the lung vessel markings in full inspiration and expiration. In forced expiration they almost entirely disappear. This observation teaches the lesson that the visibility of the lung markings is more a matter of contrast between them and the surrounding lung than of any intrinsic variation in density of the shadows themselves, and should emphasise the need for caution in making such a diagnosis as "fibrosis" or "inflammatory thickening of the lung stroma" from a radiogram.

The chest is now gone over in detail, with the tube diaphragm closed down to a suitable size in order to gain contrast. A transverse slit is useful to compare the air entry into opposite sides of the chest. The apices are again scrutinised with deep breathing and coughing. The sternomastoid muscle should be displaced from the apex with the finger or a suitable palpator. In the middle parts of the lung field it may be necessary to displace the breast or the pectoral muscle with the hand, and a pendulous breast must always be lifted when the lower lung fields are under consideration. If fluid is suspected, the patient must be tilted from side to side in order

see whether the fluid line remains horizontal and to differentiate from ribs or diaphragm.

If at this stage an abnormal shadow has been noted in the lung field or mediastinum, the examination is now directed to its localisation. The following procedures are universally employed :

Parallax Observations.—The patient is rotated about his vertical axis, and note is taken of the direction and rate of movement of the shadow or shadows relative to the normal structures, e.g. ribs, heart, and spine, and to one another. The ability to estimate the depth at which the shadows lie comes very quickly with practice. An alternative procedure, less often used, is to make the patient lean forwards or backwards alternately. This is useful in examining the apical regions ; the movement of the shadow relative to the clavicles and ribs gives a fairly accurate idea of its depth.

The Oblique Positions.—The patient is now rotated into one of the four standard oblique positions, which take their names from the position of the shoulders

(1) **RIGHT ANTERIOR OBLIQUE** ; right shoulder anterior, touching the screen or film.

(2) **LEFT ANTERIOR OBLIQUE** , left shoulder anterior, touching the screen or film

(3) **LEFT POSTERIOR OBLIQUE** ; left shoulder in contact with the screen.

(4) **RIGHT POSTERIOR OBLIQUE** ; right shoulder in contact with the screen.

The posterior oblique views are valuable in lower lobe lesions, especially if atelectasis is present, since they disengage the shadow from that of the heart.

The exact angle to which the patient is to be turned depends upon circumstances. The variations in the normal are so great that the correct angle for showing the heart's contours and the retrocardiac or posterior mediastinal clear space will be different for each patient, and must be found by observation. For a detailed description of the cardiac contours in the oblique views the reader is referred to the section on Radiology of the Heart.

Films are taken when the most satisfactory position has been found.

The Full Lateral Position.—The patient stands with the side to be examined towards the screen, and holds the arms fully extended above the head. This position should on no account be omitted. It is most valuable in showing the general disposition of the lobes and interlobar fissures, and therefore in localising a lesion to its correct lobe or segment of a lobe. It is, on the whole, the most useful view to supplement the standard postero-anterior view, since it carries into the field of chest radiography the principle of "two views at right angles to one another," which is acknowledged in all other fields of radiography to be essential. In this connection Chapters V and VI on The Anatomy of the Lobes and of the Bronchi should be studied.

Lateral View of the Lung Apices.—The apical regions are not well shown in the ordinary lateral view, because the shoulders obscure them. The

writer has described a method of avoiding this difficulty. The patient stands with the side to be examined towards the screen and raises this arm above the head. The other arm is lowered to the side. The patient then leans sideways towards the tube. The central ray enters above the clavicle and emerges through the axilla of the side to be examined. The nearer apex is thus projected above the other and not superimposed upon it. This enables the antero-posterior depth of an apical lesion to be estimated with considerable accuracy.

Oblique Illumination of the Interlobar Fissures.—The main interlobar fissures slope steeply downwards and forwards. Lesions which extend along these fissures, such as interlobar effusion, a pleural thickening, or an inflammatory process of the adjoining lung, may be more clearly seen if the ray is directed along the plane of the fissure. This may be done by elevating the tube, while the patient faces the screen, or more simply by making the patient lean backwards into the hollow-back or "lordotic" position (Fig. 299).

Examination of the Lateral and Posterior Costophrenic Sinuses.—These are examined while the patient is slowly rotated from the anterior to the lateral position. The posterior lower thirds of the lower lobes, which in the posterior costophrenic angle lie behind the summit of the diaphragm, may be shown in an antero-posterior projection by raising the tube to the level of the patient's chin and directing the ray downwards and backwards. A somewhat similar projection, due to *Albers-Schönberg*, is used to show the supraclavicular apex. It is usually carried out as a radiographic measure, with the patient lying on the back. It has the effect of throwing the shadows of the clavicles downwards. During screening, a similar effect is obtained if the patient leans forwards in the facing position, the tube being centred over the apices behind.

Tangential View.—Lesions lying near the surface of the lung—for example, pleural thickenings or localised collections of fluid, pleural calcification, osteomyelitic and tuberculous abscess of the ribs—should be examined in tangential projection, the patient being slowly rotated until the central ray grazes the surface at the point to be examined. This method is used to differentiate lesions of the chest wall and pleura from those of the lung and in estimating the depth of the lesion. It should be combined with careful observation of the relative movements of the ribs and of the shadow with respiration. In addition, care should be taken to exclude bony tumours of the scapula, e.g. exostosis, by making the patient raise the arm, so that the scapula moves relatively to the chest wall.

Examination of Young Children.—Young children are easily upset by screening, and it is better to reverse the usual order and to begin with radiography (postero-anterior and lateral films). The screening should then be carried out in spells, turning the room light on after each short spell. If the child is talked to in a quiet, matter-of-fact way during the screening, and given something definite to do, it soon conquers its nervousness. Children

below the age of 5 do not understand instructions to breathe deeply or to hold the breath, but if told to "blow," they obey at once, and a good respiratory excursion of the diaphragm and good inflation of the lungs result. If they are told and shown how to "puff the cheeks out," they soon learn how to do it. By this action the chest is inflated and the respiratory movement arrested. The film is taken at this instant. Infants are usually examined in the recumbent position, or may be held upright with the arms above the head. If they are crying, the film should be taken during the expiratory phase, when the diaphragm is moving relatively slowly. Various types of apparatus have been devised for holding an infant erect in a suspension frame, but they do not appear to have found general favour. Such apparatus is described and illustrated by *Engel and Schall*, and by *Wood*.

RADIOGRAPHIC TECHNIQUE

The normal postero-anterior radiogram is taken with the patient erect, facing the film. The shoulders should be equidistant from the film and the clavicles parallel with it. The arms may embrace the film-holder, and this tends to throw the scapulæ off the lung fields; this object is even better obtained by making the patient stand with arms "akinbo," the backs of the hands on hips and the elbows forwards. Exposure is made in moderately deep inspiration.

Technical Factors

DISTANCE.—Though a 6-foot distance is essential for showing the heart without appreciable distortion, it need not be insisted upon for chest radiography. It has the advantage of placing the entire chest in most cases on a 15×12 film, but if it necessitates an unduly long exposure, it should be abandoned. When using apparatus of only moderate power, it is better to reduce the distance to $4\frac{1}{2}$ or even 4 feet in order to obtain short exposure. Such radiograms are quite satisfactory from a diagnostic point of view. Distances below 4 feet are less satisfactory in practice.

TIME.—The technical factors should be such as to produce an exposure in less than a quarter of a second; exposures of one-eighth of a second or less are preferable. Below one-eighth of a second there is very little appreciable improvement in the quality of the radiogram.

PENETRATION AND MILLIAMPERAGE—With the development of apparatus capable of giving 200 or more milliamperes at approximately 50 kilovolts, the modern trend has been to take a very "soft" type of film. There is a tendency to overdo this softness of penetration and to produce films which are under-penetrated and under-exposed in the hope of showing very delicate lung detail or early pathological lesions. The writer believes that nothing is gained by so doing, and that a more fully exposed film is preferable. In very

soft films fine detail is more liable to be obscured by the ribs, and under-exposed films show a veiling of the lung detail by the soft parts, particularly in the apical and upper axillary regions, which is objectionable. If the experiment be made of definitely over-exposing a film of a lung showing very fine pathological shadows and of viewing it by strong transmitted light, it will be found that a considerable degree of over-exposure is tolerable before the shadows in question are rendered invisible. A seriously under-exposed film, on the other hand, cannot be made to show them whatever method of illumination is used.

When dealing with gross opacities and especially with mediastinal lesions, aneurysms, bronchial carcinomata, and similar conditions, considerable increase in penetration is essential, in order to show the trachea and to differentiate the densities composing the central shadow. A Potter-Bucky or Lysholm grid must be used in many such cases.

Stereoscopy

Stereoscopic examination of the lungs has enjoyed, and still enjoys, a greater vogue abroad than in this country. In some clinics it is employed as a routine measure. The writer's experience is that, in dealing with the gross lesions of the lung and pleura, far more information is obtained by the usual method of examination in many planes, and that stereoscopy is disappointing. In the case of the finer infiltrations of the lung parenchyma it may be very useful. Fine mottlings or patches of tuberculous exudate, bronchiectatic cavities, emphysematous blebs and bullæ, and fine adhesions in pneumothorax can sometimes be more clearly visualised and more easily localised by this method. Expense can often be reduced by using a pair of small films for stereoscopic examination of special regions, or for "sample" stereograms of small areas. The cassette-holder in the writer's apparatus is provided with a set of adapters to take all the smaller sizes of film. When opaque material, such as lipiodol, has been injected into sinuses or into the bronchi, the stereoscopic pictures bear comparison with those obtained in the skull and other bony parts, and are of undoubted value. In general, however, the reluctance of most radiologists to incur the additional expense of routine stereoscopic films is fully justified by experience.

LUNG TOMOGRAPHY

Tomography (Planigraphy, Stratigraphy) is a recently developed method of radiography which shows one selected plane or stratum of the body clearly defined, while blurring the shadows of all structures lying outside that plane. This is accomplished by moving the tube and film simultaneously in opposite directions, the tube and the film-carriers being coupled together by a pendulum or other means. Such apparatus has been developed by *Bocage* (1921), *Ziedses des Plantes* (1921-1925), *Vallebona* (1933),

Grossmann, and by the writer. *Grossmann's* apparatus consists of a pendulum carrying the tube at its upper end, and the film-carrier at its lower end. The axis about which the pendulum rotates can be raised or lowered until it is in the same horizontal plane as the selected stratum in the patient's body. The patient is lying horizontally during the exposure, which may be from one half-second up to several seconds. *Grossmann* has described his apparatus and the principle involved in the *British Journal of Radiology*.

The writer has described a simple attachment enabling tomography to be carried out on any X-ray couch of the usual pattern: the tube and the Potter-Bucky carrier are coupled together by a simple lever, which transmits the motion of the tube through connecting rods, reciprocally to the carriage. The desired stratum is selected by varying the position of the axis along the lever. The same principle, or a simple slotted lever, can be adapted to a vertical radiographic stand for carrying out chest tomography in the erect position.

Applications of Tomography to Chest Examinations.—These have been principally studied abroad by *Chaoul* and *Greineder*, and in this country by *McDougall* and by the writer.

(1) **NORMAL LUNG ANATOMY.**—A study of the anatomy of the pulmonary vessels and of the bronchi has been made by *Greineder*. A series of tomograms through many planes allows the course of the vessels to be followed from their origin to their termination and demonstrates their relations to one another. They stand out clearly in the lung field, undisturbed by the shadows of the ribs or of the lung parenchyma. The arteries branch out freely from the main pulmonary arteries from the upper hilum, the veins lie to the outer side and enter the left auricle posteriorly at a lower level. The clinical value of this is that it facilitates the study of lobar anatomy in lobar lesions, since the vessels going to the different lobes can be identified, and their displacements by fibrosis or atelectasis of individual lobes can be more readily observed than in plain radiograms. (See Figs. 87 and 94.)

(2) **CAVITIES.**—The method shows very clearly cavities invisible in ordinary radiograms, because it increases the contrast between the structures in the selected plane, which are held on the film throughout the exposure, and structures in other planes, which are blurred and diminished in density by the movement. Two kinds of cavities may thus be brought into prominence: (a) *Thin-walled cavities*, in tuberculous disease, which are surrounded by normal or mottled lung fields, and (b) *thick-walled cavities*, or areas of softening and caseation, surrounded by dense consolidations, which are normally either difficult to be sure of or actually invisible. Not only may the existence of the cavities be thus proved, but valuable information as to the character of their walls may be obtained. Many abscess cavities which in plain radiograms have shown merely a vague translucent area with a fluid level are proved by tomography to be multi-locular, or honeycomb-like, or to be partially divided by septa projecting into their lumen. Portions of necrotic lung tissue lying

within abscess cavities can be shown. Nodulation of the wall of the cavity is sometimes a prominent feature in tomograms of malignant abscesses. The writer has on several occasions succeeded in tracing the drainage bronchus into an abscess cavity in a tomogram, though the corresponding plain radiogram had failed to show it. If the bronchus, on the other hand, has been



FIG. 73.—Right pneumothorax. Doubtful cavity in right upper lobe.

Tomogram of same case showing cavity clearly.

obstructed by growth, the tomogram shows this very clearly without the use of lipiodol. Even the presence of a large effusion, causing complete opacity around the neoplastic lung, or gross atelectasis, is not an insuperable obstacle. The tomograph has in the writer's hands been of distinct value in showing bronchial obstruction in such cases.

THE LUNG PARENCHYMA

The lung lobules are marked out on the surface of the lung in polyhedral areas of from one-quarter to three-quarters of an inch in diameter, bounded by lines of pigment. Within the lung the lobules are roughly pyramidal in shape and are separated by connective tissue septa which tend to limit the spread of infection from one lobule to another, thus accounting for the fan-like densities often observed radiographically. Fig. 75 shows well the lobules on the surface of the lung in the middle lobe.

Internal Structure of the Lobule.—This has been described by *Laguesse* (1899). At the apex of the lobule there enters a sublobular bronchus, which runs through the middle of the lobule to a point near its centre, where it



FIG 74 Bronchial carcinoma marked by pleural effusion



Tonogram of the same case, showing complete stenosis of the right bronchus (arrow)

divides into two intralobular bronchi, giving off a number of collateral bronchi before dividing, which supply the upper part of the lobule. The two main branches subdivide repeatedly and give rise to a number of small terminal bronchioles. Each of these ends in a cluster of short branches bearing numerous alveoli. Each cluster is an acinus, so called from its resemblance to a bunch of grapes. (Fig. 76.)

Internal Structure of an Acinus.—Each terminal bronchiole, about half a millimetre in diameter, divides into respiratory bronchioles, which have

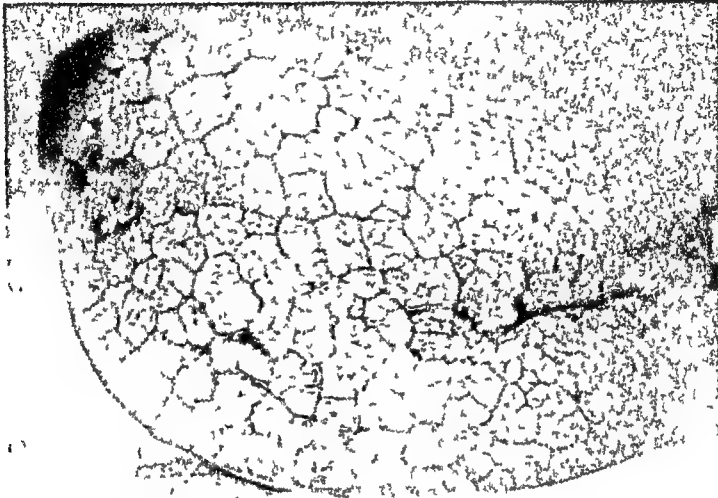


FIG. 75.—Middle lobe of the lung—inflated. To show lobules in surface: the polygonal areas, outlined by pigment, are the bases of the pyramidal lobules. Half actual size.

alveoli on their walls, opening into the lumen. After a very short course of 1 or 2 mm. they may again subdivide into respiratory or "transitional" bronchioles of the second order, or into alveolar passages. There are slightly longer passages devoid of cilia, but thickly beset with alveoli. These give off alveolar sacs, which have a diameter varying from one-fifth of a millimetre in the child to three-quarters of a millimetre in the adult; their walls consist of many alveoli. According to *W. S. Miller*, the alveolar sacs arise from the alveolar passages (or "alveolar ducts") through an intermediate stage of "atria." Several atria are attached to each duct, and three to five alveolar sacs to each atrium. A complex of atria, alveolar sacs, and alveoli he terms a "primary lobule" (see Fig. 171). The minutiae of these histological subdivisions do not concern the radiologist, since they are too fine to be observed in a radiogram of the lung. The conception of the acinus has, however, found its way into the radiological literature, especially in Germany, and, since the acinus is a macroscopic entity, it is of some importance. *Loeschke* described the acinus as that portion of the lung supplied by the true terminal bronchus. This u

of lung parenchyma is about one-quarter inch in diameter. *Husten's* acinus is a somewhat smaller unit, the respiratory bronchiole of the first order with all its branches, while *Grellmann* restricts the term to the system of alveolar passages and alveolar ducts: his acinus is about one-eighth of an inch in diameter. *Aschoff* believes that the narrow point of the system of small air passages is in the first respiratory bronchiole, and that micro-organisms tend to be arrested at this point; the resulting lesions spread to the segment

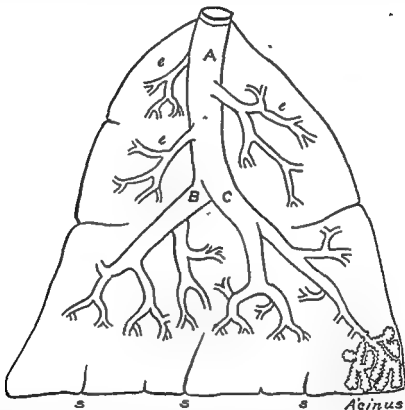


FIG. 76.—Diagram. Lobule of lung and acinus $\times 8$ (*Laguesse*). The lobule has a base of about 13 mm. Capacity 1 c.c. A, intralobular bronchiole. B, C, its main divisions. e, e, collateral branches. s, s, s, septa limiting the subdivisions of the lobule.

supplied by them. In this way arise small infiltrations which have rosette-like appearance. These are referred to as "acino-nodose" lesions by German writers. *Graeff* and *Kupferle* have made a comprehensive study of such lesions by comparing radiograms with post-mortem specimens in the same cases.

In order to gain some idea of the arrangement and dimensions of the terminal portions of the lung parenchyma which might serve as a basis for the radiological interpretation, the writer has made a considerable number of

injections of lipiodol into inflated lungs and has studied these by stereoscopy and by photographic enlargement. One of these specimens is illustrated in Fig. 79. In the upper part of the specimen only the medium-sized bronchi are

filled. In the next section are seen medium-sized bronchi with a few collateral bronchioles of a $\frac{1}{2}$ to 1 mm. in diameter. Below this is a section in which the respiratory bronchioles are commencing to fill, the general appearance resembling a tree in bud. Attention is drawn to the upper part of this section, which gives the bronchial supply of an entire lobule. It reproduces almost exactly the branchings seen in the diagram (Fig. 76) after *Laguesse*. In the lower part of the photograph the alveolar passages and some of the terminal alveoli are filled. The parenchyma here is divided up into units of about one-eighth

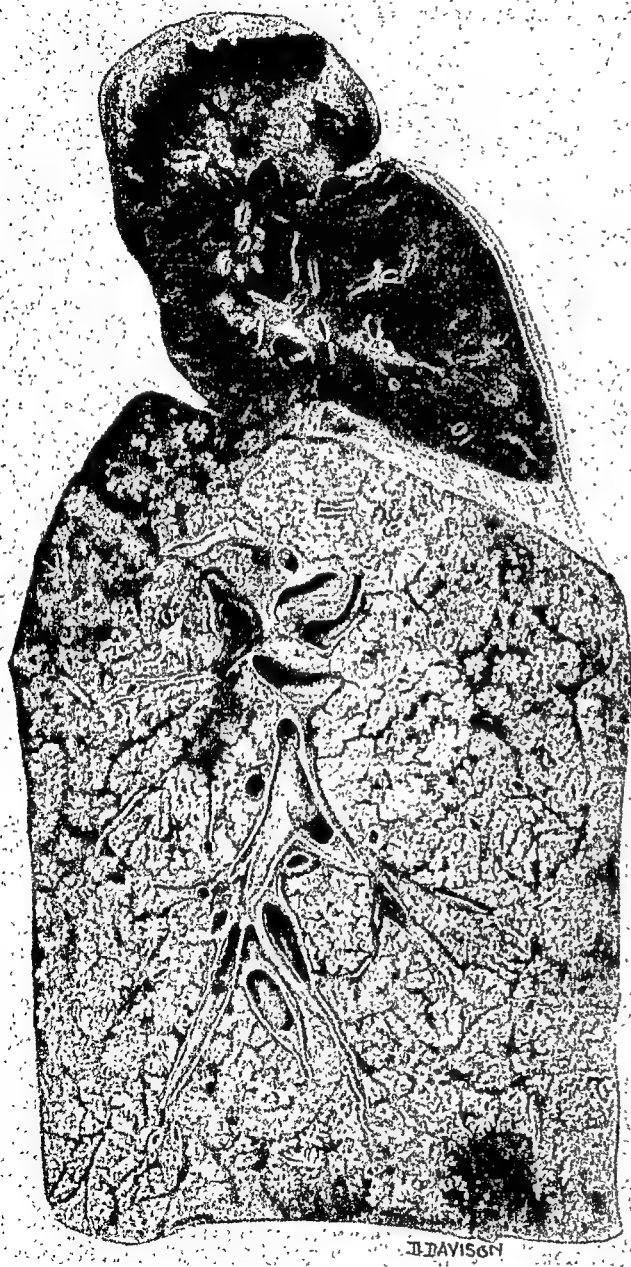


FIG. 77.—Specimen: Pulmonary phthisis. In the lower lobe multiple lesions, showing rosette-like “acinous” groupings in the distribution of the terminal bronchioles.



FIG. 78.—A small portion of the same specimen as Fig. 77 slightly magnified, to show rosette-forms.



Medium bronchi.

Medium bronchi and
bronchioles

Terminal and respira-
tory bronchioles.

Alveolar passages and
alveoli, showing
grouping into sub-
lobules with rattle-
like arrangement

FIG. 79 Specimen Study of lung parenchyma, after lipiodol injection, showing from above downwards successive degrees of filling of the finer lung detail. Inflated human middle lobe, post-mortem specimen. Enlarged about $\times 1\frac{1}{2}$ times. Author's preparation.

of an inch in diameter, each of which represents an acinus of *Husten*, very nearly, these units being usually grouped together in sets of four. Owing to the divergence of the respiratory bronchioles at right angles to one another, these groups are frequently found in radiograms in the form of a rosette, especially when they lie near the surface of the lung and are viewed in the axis of the terminal bronchiole. The individual petals of the rosette sometimes form a solid shadow, but may take the form of hollow rings giving rise to the "breadcrumb" mottling of some chronic tuberculous lesions. The rosette-like appearance is well brought out in Fig. 77, which is a drawing from a specimen of acute tuberculous broncho-pneumonia.

Similar rosette-like markings are sometimes seen in miliary carcinomatosis, broncho-pneumonia, pneumoconiosis, and in emphysematous areas of lung near the surface in miliary tuberculosis, chronic emphysema, and silicosis.

It is somewhat unfortunate that so much confusion exists at the present time in the nomenclature and description of the finer details of the lung parenchyma. Readers who desire to obtain a more complete knowledge of the subject should consult *W. S. Miller's* book, already referred to, and the discussion of the subject by *Grethmann*.

CHAPTER XII

THE CHEST WALLS

THE SHADOWS on a chest film should be studied in some definite order, for example: (1) chest walls (bony framework and soft parts); (2) diaphragm; (3) median shadows; (4) hila; (5) lungs and pleura.

THE CLAVICLES, STERNUM, SCAPULÆ, AND SPINE

Clavicles.—The sternal ends should be equidistant from the midline; if not, the radiogram is not perfectly positioned or the chest is asymmetrical. A fine line along the upper border of clavicle is due to skin. The clavicles are absent, defective, or divided in cleido-cranial dysostosis.

Sternum.—The sternum is merged in the median shadow. As a rule, only the upper, and occasionally the lateral, borders of the manubrium are visible in the postero-anterior film. The gladiolus does not show, unless the patient is rotated slightly to one side, when its notched border may become visible. Deformity of the sternum may be noted in pigeon-chested individuals, and in certain trades, e.g. shoemakers, in whom a deep depression of the lower sternum may be found in the lateral view. In emphysema the sternum may be markedly bowed forwards.

In the lateral view erosions of the sternum are best shown; usual causes are secondary carcinoma of the breast, tuberculosis, and osteomyelitis. Erosion of the sternum from within the chest may be caused by malignant tumours of the thymus and other mediastinal tumours, gumma, and aneurysm.

Scapulæ.—The shadows of the scapulæ do not give rise to any difficulty in the postero-anterior film. This should always be taken in such a way as to throw them on to the outer part of the chest, either by directing the patient to put his arms around the film-holder, or to stand with hands on the hips and the elbows rotated forwards into contact with the film support.

In the lateral view the shadows of the scapulæ are often troublesome. In this view the dense axillary borders of both scapulæ are seen crossing the upper posterior part of the chest, and often puzzle the less experienced observer.

Spine.—The spine is merged in the median shadow; the individual vertebræ in properly exposed negatives are nearly or quite invisible. The intervertebral spaces may just show. Scoliosis produces a projection of the spine to one side of the median shadow, often mistaken for tumour of the mediastinum. This applies also to the spindle-shaped or circular spinal abscess of Pott's disease. In the soft radiograms necessary for chest work, the bone detail does not show, and such shadows *seem* to be part of the mediastinal shadow.

THE RIBS

The ribs should slope symmetrically on the two sides. In scoliotic chests they fall more steeply on the side of the concavity. In Pott's disease, with vertebral collapse, they are closely spaced and convergent. Apart from spinal deformity, steeply sloping ribs with narrowed intercostal spaces indicate diminished lung volume on that side, from old or recent disease. The ribs are more horizontal, and the spaces widened in emphysema, pneumothorax, and large effusions. Anteriorly the ribs end well outside the midline.

The cartilages, healthy or diseased, are invisible, unless calcified. Calcification in costal cartilages is common and not pathological. Dots of calcification in lower costal cartilages slope upwards in line with the descending shadows from the hila, and may be mistaken for calcification in hilar glands. The first rib cartilage is the first to calcify, and the shadow may easily be mistaken for a calcified tuberculous apical lesion.

Developmental Abnormalities of Ribs.—These occur in 1 per cent. of chest films. Cervical rib (bilateral in 70 per cent. of cases) usually occurs on the seventh cervical vertebra, but has been noted on others. It is seen in 0.4 per cent. of chest films, twice as often in women as in men. It varies in size from a small element fused with, or articulating with, the transverse process, to a completely formed rib. It may fuse or articulate with the first rib, or have a free extremity. It sometimes consists of two articulated portions. If it has a free end it is usually continued by a fibrous band, the point of attachment of which is often marked by a small elevation on the first rib near its costochondral junction. If a large cervical rib is present, the first rib may join the sternum at a lower point than normally. The first thoracic rib may have the characters of a cervical rib on one or both sides: a complete count of the cervical vertebrae is necessary to decide the point.

Fusion or articulation of the first two ribs with one another may occur. The variations are legion. In the other ribs many congenital abnormalities occur, such as: (1) absent or supernumerary ribs, with interpolated half-vertebrae, congenital scoliosis, and elevation of the scapula; (2) fusion or articulation of two or more ribs; (3) splaying of the anterior ends; (4) forking of the anterior ends; the space enclosed within the fork, projected on the chest, may simulate a cavity in the lung.

Diseases of the Ribs.—Many diseases of ribs may be noted in the course of a chest examination, such as:

RICKETS.—Cupping and broadening of anterior ends: Harrison's sulcus (shown as a slight concavity of the parietal walls).

ACHONDROPLASIA.—The ribs are coarse and broad, and abnormally curved.

OSTEOGENESIS IMPERFECTA.—Abnormal curvature of the ribs and fracture.

PAGET'S DISEASE.—The ribs are abnormally dense. Sometimes only a few ribs, or even a single one, are affected.

ALBERS-SCHONBERG'S DISEASE.—This usually affects all the ribs, which are extremely dense.

General or localised Rarefaction of the ribs occurs in (1) old age (senile osteoporosis); (2) fibrocystic disease; (3) multiple myelomatosis; (4) secondary carcinoma; and (5) osteomalacia.

Local Deformity or Nodular Enlargement of ribs occurs after fractures and after operation for empyema. In the latter case the defect may be closed by bridges of bone joining the upper and lower margins, enclosing a rounded space, originally made by the drainage tube. This should not be mistaken for a cavity in lung.

Localised Erosions or Expansion of Ribs may be found in bone disease, primary or secondary, of the ribs themselves: sarcoma, carcinoma, hæmangio-endothelioma, gumma, chondroma, osteoma, cyst, osteomyelitis, tuberculosis.

Rib Changes in Intrathoracic Disease.—From the point of view of intrathoracic pathology, the following should be specially noted:

(1) **NOTCHING OF THE LOWER MARGINS OF THE RIBS**, in coarctation of the aorta. The upper two or three ribs usually escape: the fourth to the eighth are usually affected. The appearance of multiple shallow notches, due to convoluted enlarged intercostal arteries, is distinctive, and in several instances has been the first sign leading to diagnosis of the condition. (Roesler's sign.)

(2) **LOCALISED EROSION OF BONE**, of an invasive character. This is useful in the differential diagnosis of malignant disease of the lung. It may result (a) from direct invasion of rib, via the pleura, by a carcinoma or other malignant tumour of the lung or pleura, or (b) by lymphatic spread from the mediastinum to the intercostal lymphatics, in bronchial carcinoma or malignant disease of the mediastinum.

The sign is also useful in the differentiation of actinomycosis (and other severe mycoses) from other inflammatory lesions. Actinomycosis tends to invade the ribs; gumma may do so. Tuberculosis of the lungs hardly ever spreads to the rib, but a tuberculous abscess of a rib, accompanied by erosion, may simulate a tumour of the pleura.

The posterior ends of the ribs may be eroded, and the rib space widened, by neurinoma arising from the spinal nerves (hour-glass tumour).

Erosion of the first ribs may result from any malignant tumour at the apex of the lung.

THE SOFT TISSUES OF THE CHEST WALL

The Female Breasts cast marked shadows, and when pendulous may completely obscure the base. When one breast has been removed, the obscurity caused by the other simulates disease and is a certain trap for the unwary. The nipple shadow is often seen. A shrunken carcinomatous breast produces an opacity resembling a lung tumour. At the apex unilateral obscurity may result

from an excess of fat, enlarged or calcified cervical glands, soft tissue swelling in the neck, or thyroid enlargement; diminished density, with a mottled appearance, from subcutaneous or intramuscular emphysema.

Muscles.—The pectorals veil the upper chest and may be asymmetrically developed, causing a difference of translucency on the two sides. The sterno-mastoid often obscures one apex, especially if the head is turned towards that side.

Soft Tissue Shadows at the Apex.—There are several shadows often found at the normal apex which may puzzle the beginner. The sterno-mastoid muscle, with its sharp outer edge, obscures the inner part of the apex. Its edge traced downwards becomes continuous with a fine line parallel with the upper border of the clavicle, due to the skin dipping into the supraclavicular fossa.

The "companion shadows" of the first and second ribs are faint shadowssometimes found along the lower borders of these ribs. They have given rise to a good deal of speculation. They are due to the extrapleural tissues, and their appearance or non-appearance depends simply on whether the apex of the lung is projected on to a space or on to a rib. The

relation of the apex of the lung to the lower borders of the first and second ribs is variable, partly as a result of anatomical differences (e.g. kyphosis) and partly owing to varying angles of projection. *Knutsson* believes the shadows accompanying the second rib to be due to a special fold of pleura ("Knutsson's sheath"), containing the superior intercostal artery and veins, the inferior cervical ganglion of the sympathetic and the first dorsal nerve (Fig. 80). These relationships are of importance, since they explain the neurological signs (first dorsal nerve involvement and Horner's syndrome) which may be found in tumours of the thoracic inlet. This space forms the medial part of the com-

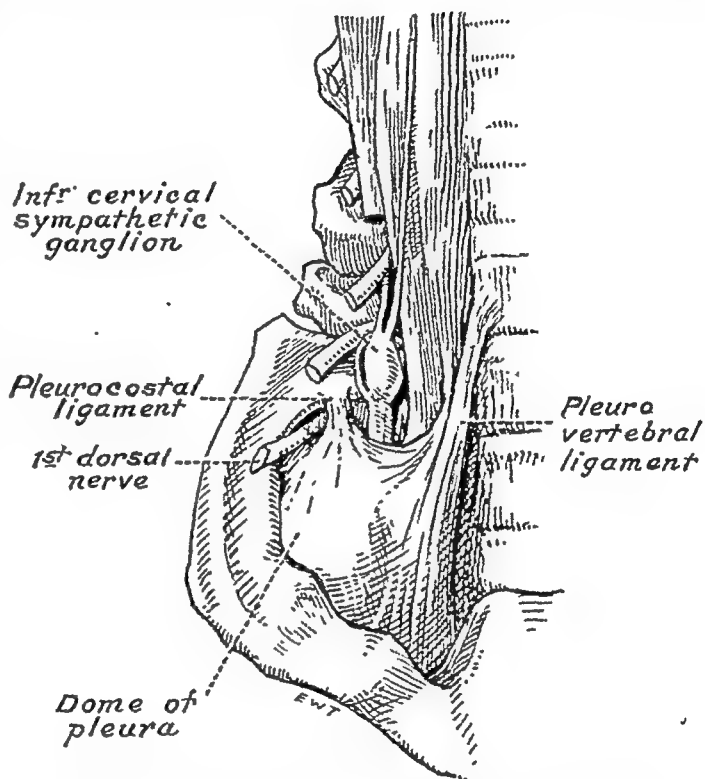


FIG. 80.—Diagram: Relations and attachments of the apical pleura (after Poirier). (*Traité d'Anatomie Humaine.*)

panion shadow of the second rib. The pleural dome is closely bound to the first rib on the outer side of this. It is also fixed to the spine, and to the first rib by the pleurovertebral and the pleurocostal ligaments.

Careful stereoscopic radiographic and anatomical study by *Bönniger* shows that the apical dome of the pleura is usually more posterior than described by anatomists, often below the posterior end of the second rib and behind the posterior part of the first rib. The highest part of the apex of the lung does not, therefore, lie in the thoracic aperture.

The companion shadow of the second rib crosses the first rib and can be traced along the lateral thoracic wall as far as the third or fourth rib. Lower down it disappears; but if the patient is turned so that the rays are tangential to the postero-lateral chest wall, a shadow is again seen on the inner side of the ribs, extending from rib to rib and clothing their inner surfaces; it is due to the internal intercostal muscles, which in muscular subjects almost completely cover the inner surfaces of the posterior ribs (*Knutsson*). On the lateral and anterior parts of the chest, the greater part of the inner surfaces of the ribs is not covered by intercostal muscle, but is bare; this explains why the companion shadow of the lower ribs is invisible in normal anterior views, in which only the lateral walls are tangential to the rays.

It often happens that the companion shadow is seen on one side only. This results from retraction of the lung apex, and may occur in any condition causing diminished lung volume; the writer has noticed it in bronchial carcinoma with bronchostenosis, pleural effusion, upper lobe consolidation, delayed resolution of pneumonia and pulmonary tuberculosis. It is a mistake to diagnose thickening of the apical pleura whenever this shadow is seen, although such thickening is often present in association with apical tuberculosis and fibrosis. In the latter case the margin of the lung is usually crinated and puckered. In most instances of widened "companion shadow" the two layers of pleura are actually in apposition and nothing intervenes between them. Although a pleural effusion may run right up the chest wall and increase the depth of the companion shadow by arching over the apex, apical encysted effusion is rare.

Bönniger has also shown that there is another companion shadow of the first rib. This is a faint shadow seen on the inner side of the descending part of the first rib, which it never crosses. The shadow tapers to a point when traced downwards, and ends where the pleura becomes attached closely to the rib. *Bönniger* explains the shadow as follows: stereoscopic studies which he has made, both in the living patient and in the cadaver, show that the anterior pleural dome is separated from the posterior part of the dome by a slight furrow or groove. The point at which the images of these two domes overlap one another is sometimes marked in a radiogram by a slight "nick." Between the anterior dome and the first rib lies the companion shadow of the first rib (*Bönniger*). Between the posterior dome and the second rib lies the companion shadow of the second rib.

SUBCLAVIAN ARTERY.—If the left border of the mediastinal shadow is traced upwards towards the apex, it arches outwards above the clavicle and gradually fades out. *Assmann* has shown by injection that this arching shadow corresponds with the position of the left subclavian artery. The shadow is due, not so much to the artery itself, but to the opacity caused by the indentation of the lung by the subclavian groove. This groove is deepest at the mediastinal edge of the lung ; traced outwards, it rapidly becomes narrower and disappears as the artery and vein pass outwards on to the first rib. This explains the fading out of the subclavian shadow when traced outwards. The groove varies in depth in different subjects, so that the shadow is variable in density and often absent.

CHAPTER XIII

THE DIAPHRAGM

IN POSTERO-ANTERIOR projection the normal diaphragm shows a well-defined bowline, convex upwards. The upper contour, variable in relation to the ribs (fourth to seventh costochondral junction), represents the highest part of the diaphragm, normally the anterior half or anterior third. Owing to the slope of the diaphragm downwards and backwards, the contour shown in any given radiogram will vary with the angle of inclination of the rays, being merely that part to which the rays are tangential. "Diaphragm movement" is usually assessed by noting the degree of movement of the bow during postero-anterior screening, and serves as a rough guide to diaphragmatic mobility, though what is actually being observed is the movement of various parts of the diaphragm, as they successively become tangential to the rays. For complete examination, screening in the lateral and both oblique views must be carried out in addition. The lateral view may show the posterior halves to be moving freely, though the summit moves but little.

Localised limitation of movement, or *localised* elevations and fixations of the diaphragm, should be looked for in these projections.

ANATOMICAL VARIATIONS OF CONTOUR

On the right side the contour of the diaphragm may be divided into two or more arches, the outlines of which appear to cross one another (Fig. 81). When there are two such arches, the inner is higher than the outer. It has been shown by *Thomas* that the former is antero median, and corresponds to that part of the diaphragm in the neighbourhood of the inferior vena cava and right heart border, which limit, to some extent, its downward movement. He has also shown that it is separated from the rest of the diaphragm by a shallow groove, due to contraction of the largest and strongest diaphragmatic muscle fibres which run obliquely outwards and forwards from the vertebral column to the central tendon, and, in a continuation of the same line, by those which reach the antero-lateral part of the tendon from the chest wall (eighth and ninth ribs). The diaphragmatic muscle fibres covering the antero-median elevation are relatively short and weak. The groove becomes less obvious, or disappears, when the diaphragmatic contraction relaxes in expiration. The anatomical basis of this variation is described in detail by *Assmann*.

The antero-median dome thus produced sometimes projects strongly as a rounded elevation into the thorax, and may be mistaken for a tumour or cyst of

the liver. It is, however, exceedingly rare for a tumour to project in this way. Tumours or cysts of the liver usually exert pressure in all directions on the semi-fluid liver substance, with the result that the liver is enlarged as a whole,

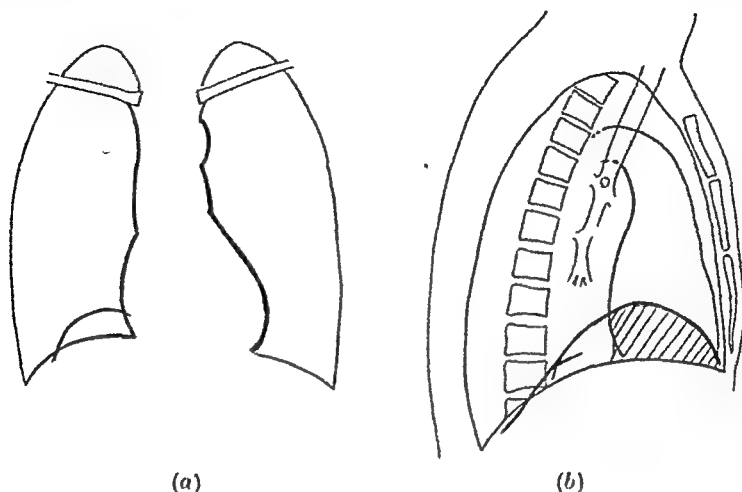


FIG. 81.—Diagram: (a) Dome-shaped elevation of medial half of diaphragm. (See text.) (b) Lateral view. Superimposition of this upon the heart shadow in the lateral view may be mistaken for an interlobar effusion.

and the right diaphragm is thereby raised, even as much as 6 inches above the left, but maintains its smooth arched contour without local nodulations. If the lesion is situated on the upper surface of the liver it may, however, show a

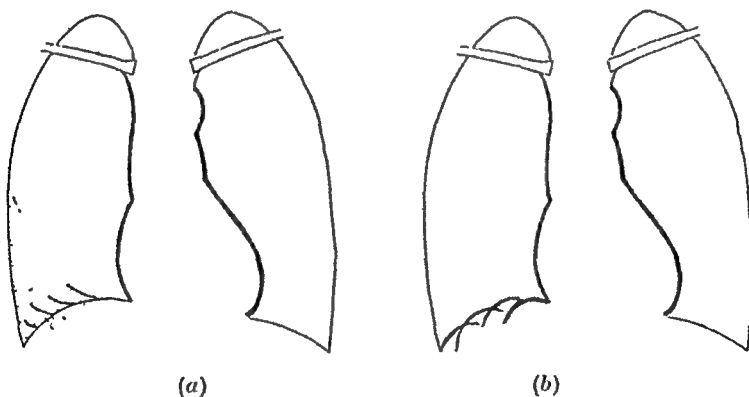


FIG. 82.—Diagram: Muscular irregularities of diaphragm contour. (a) Slips of origin from ribs; (b) irregular contraction of the fibres.

rounded local elevation. The antero-median part of the diaphragmatic dome, in the lateral view, overlaps the heart shadow, and the increased density thus produced may be mistaken for an interlobar effusion, or even a con-

solidated middle lobe (Fig. 81 (b)). The mistake could only occur if screen examination had been omitted or had been perfunctory. Occasionally several furrows are present, due to localised contractions of slips of origin of the diaphragm. The diaphragm contour is then divided into a number of small intercrossing arches. The furrows converge upon the central tendon. In some normal individuals, in fullest inspiration, and still more often in cases of emphysema, with abnormally low diaphragms, the diaphragmatic contour is divided into a number of horizontal segments, with a "step-ladder" arrangement (Fig. 82 (a)). These horizontal lines run inwards from the ribs, one from each rib, and are due to individual anterior and antero-lateral slips of origin of the diaphragm; they are normally present, but only seen when uncovered by an unusually low position of the summit of the dome. This particular irregularity is often diagnosed incorrectly as being due to adhesions.

In antero-posterior films of the chest and upper abdomen the lower limit of the posterior *cul-de-sac* is indicated by a slightly arched line some inches below the diaphragmatic dome. This line sometimes shows, near the spine on both sides, a slight elevation due to the upper poles of the kidneys, from which the pleural sacs are separated by the diaphragm.

PATHOLOGICAL VARIATIONS OF CONTOUR

The contour is often irregular. Adhesions may occur over a wide area, and break up the normal bowline into an irregular series of lines and peaks (fibrosis, pneumoconiosis). More often the peaks are few and tent-shaped. These usually occur at the lower ends of the fissures, and result from organisation of pleural exudate there. Examination at suitable angles often shows that they are continued upwards as linear thickenings in the interlobar fissures. They occur very frequently at the lower end of the main fissure, but are sometimes visible at the anterior end of the minor fissure, and in the fissure of the infracardiac lobe. Similar peaks may also be present in the pericardium extending into the fissures. Some "tentings" are thought to be due to traction of diseased and inextensible bronchi upon the diaphragm where the lung has become adherent.

Adhesions of diaphragm to the chest wall in the costophrenic *cul-de-sac* are easily recognised. The outer part of the diaphragm remains fixed during inspiration, and the costophrenic angle is obliterated.

Adhesions in the posterior *cul-de-sac* may be noted in the lateral view. The diaphragm runs upwards to meet the contour of the posterior chest wall, and the *cul-de-sac* is obliterated (Fig. 94 (b)).

As a rule these adhesions are the result of old inflammation and are symptomless. Occasionally they result from a recent diaphragmatic pleurisy and are accompanied by symptoms; lower costal pain and tenderness, and pain in the shoulder, and cough.

PATHOLOGICAL VARIATIONS IN POSITION

High Position of the Diaphragm.—The diaphragm is raised on one or both sides in many SUB-DIAPHRAGMATIC CONDITIONS, viz. Ascites, meteorism, pregnancy, abdominal tumour, cyst or abscess, enlarged spleen, renal tumour, perinephritic abscess.

THE INTRATHORACIC CAUSES are numerous. Apart from congenital abnormalities, such as failure to develop an entire lobe, the cause is nearly always to be found in an acquired diminution of volume of a lung. Atelectasis, from compression of lung, or bronchial stenosis (neoplasm, foreign body) and fibrosis (tuberculosis, chronic interstitial pneumonia) are common causes. In commencing or small effusions, and in diaphragmatic pleurisy, the diaphragm is often fixed in the expiratory position. In phrenic paralysis (poliomyelitis, peripheral neuritis, compression by mediastinal tumour, etc.) and after phrenicectomy, the diaphragm is raised and may show paradoxical movement. The elevation after phrenic evulsion is not very marked as a rule.

Localised elevations of the diaphragm may result from intrathoracic causes: In “collapsing” or “fibrosing” conditions of the anterior part of the left upper lobe, or of the middle lobe, the anterior part of the diaphragm is raised; in lower lobe lesions the posterior part. A very characteristic shape is noted in many cases of atelectasis (e.g. neoplasm of upper lobe, massive collapse), in which the diaphragm contour, usually the right, continues medially without interruption on to the right heart border, filling up the cardiophrenic angle.

Low Position of the Diaphragm.—This may be bilateral or unilateral.

(a) **BILATERAL DEPRESSION** occurs in emphysema and in visceroptosis. The diaphragm is flattened out, and the costophrenic angle approaches a right angle.

(b) **UNILATERAL DEPRESSION.**—Pneumothorax, large effusions, and over-distension of the lung (for example in valvular obstruction of a bronchus by foreign body) depress the diaphragm on one side. On the left side a flattening of the “magenblase” may be observed with a large pleural effusion.

DIAPHRAGMATIC MOVEMENTS

On quiet breathing the range of movement of the dome is normally about three-quarters of an inch. On deep breathing about 1½–2 inches; with forced breathing anything up to 3 inches may be observed.

In costal breathers the anterior attachments of the diaphragm are carried upwards by the ribs and the arch may be seen to rise, instead of falling, towards the end of a full inspiration.

Observation of the diaphragm movement in children are more easily carried out if the child be instructed to “blow” instead of to “breathe deeply.” This ensures full ventilation of the lung, since a full inspiration automatically precedes each “blow.”

Bilateral Deficiency in Movement is observed in emphysema, bilateral fibrosis, and in many of the above-mentioned conditions which produce elevation.

Unilateral Diminished Movement occurs as an early sign in pleurisy, diaphragmatic pleurisy, and subphrenic abscess, and is almost constantly found in extensive and chronic lung lesions. Lagging of one diaphragm is an expression of diminished movement. Both diaphragms actually contract together, but the more active diaphragm is able to overcome the abdominal pressure from the first, while in the diseased side the initial contraction, working against abdominal pressure and a diseased lung, is insufficient to move the tendon appreciably, so that the healthy leaflet is well on its way before the diseased one is seen to move. As *Assmann* points out, in acute conditions (e.g. pleurisy, acute cholecystitis, perinephritic abscess, peritonitis) diminished movement on the affected side is a protective reflex. In chronic conditions, such factors as pleural adhesions, bronchostenosis, and fibrosis of the lung doubtless play a part in hindering free movement of the diaphragm on the affected side. The movement of the central tendon is restricted in any condition associated with a rigid mediastinum—especially in chronic mediastinitis and pericarditis (Pick's disease).

Paradoxical Movement.—An upward movement of one leaflet of the diaphragm in inspiration occurs.

(1) From overaction of one diaphragm. Experimentally, it is observed when one phrenic is stimulated with the faradic current.

(2) In phrenic paralysis or paresis.

(3) In pneumothorax.

(4) In some cases of hernia and relaxation of the diaphragm.

The result is a "see-saw" action of the two leaves of the diaphragm. The upward movement of the affected half is demonstrable if an inspiratory effort is made with closed mouth or nostrils (as in Muller's experiment): in some cases it is brought out well by making a sudden short inspiratory sniff through the nose, with the mouth closed (mediastinal "jerking"). The principal factors in producing a paradoxical movement are. (1) weakness of the affected leaflet (phrenic paresis, or reflex inhibition); (2) diminished air entry into the lung of the same side, and (3) the decrease in intrathoracic pressure which results from inspiration.

DIAPHRAGMATIC HERNIA

This is fully described in another section. The condition may occasionally present itself as a chest lesion: when the lower part of the thorax contains air-filled hollow viscera, or when it contains a solid viscus. In the former case the diagnosis is not immediately obvious. If the hernia contains the stomach, its wall may resemble the diaphragm, while the diaphragm itself, owing to the size of the defect in it, is indistinguishable as such. If the colon or small intestine

lie in the thorax, the rounded air-filled sacculations are usually characteristic, but have to be distinguished from "cystic disease" of the lung. Barium examination, meal, and enema, may be necessary. An anterior hernia of the stomach may simulate a cyst or localised pneumothorax, and a right para-oesophageal hernia may in the postero-anterior view resemble a "fourth lobe."

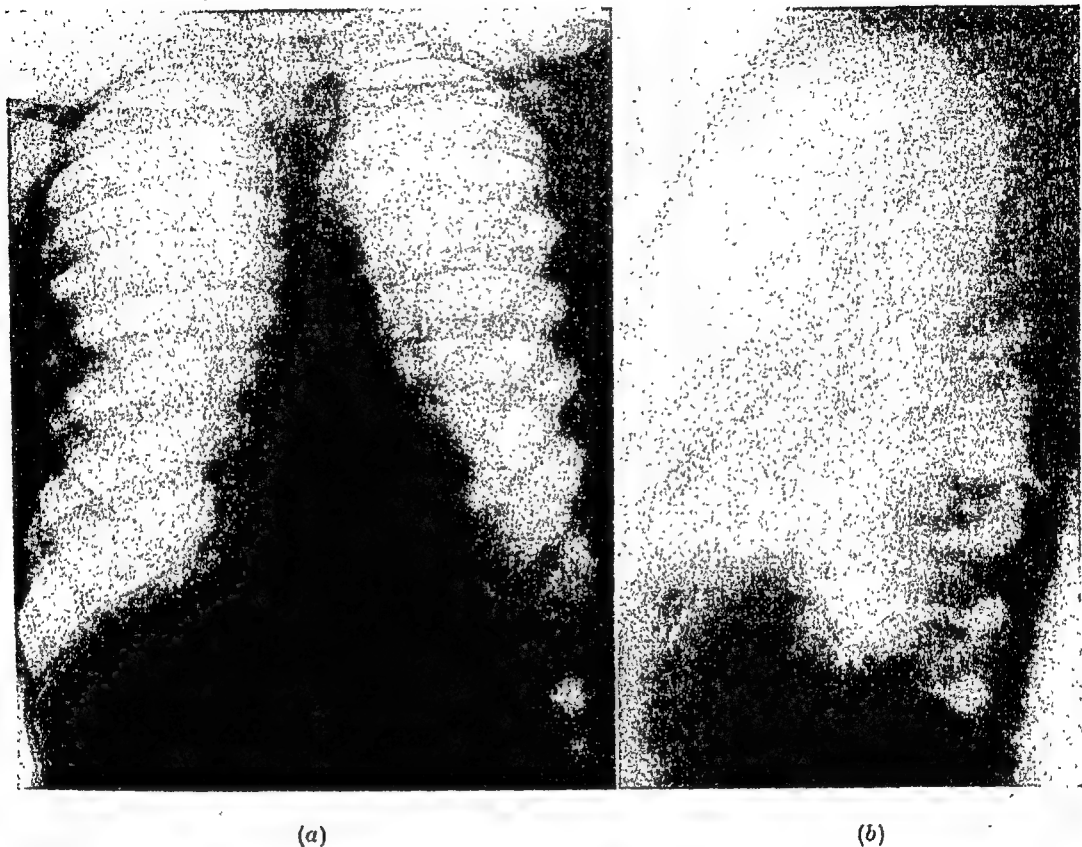


FIG. 83.—(a) Diaphragmatic hernia of spleen. Rounded shadow at left base above the diaphragm due to spleen. (b) Lateral view. Rounded shadow of herniated spleen in posterior costophrenic angle. Operation. Recovery.

In the rare instance of a solid viscus becoming prolapsed into the thorax, the appearances can be very puzzling, as in the following two cases seen by the writer.

(1) **HERNIA OF SPLEEN.**—After a fall from his bicycle, the patient, a child of 7, had abdominal pain and vomiting; and during the next two months mild febrile attacks and a troublesome cough (Fig. 83).

X-ray examination showed a circular opacity in the left hemithorax posteriorly, just above the diaphragm, with well-defined contours. The normal splenic opacity was not visible below the diaphragm. The writer gave an opinion that

it was a diaphragmatic rupture with herniation of the spleen. A small bubble of air was noted on one occasion near the shadow, thought to be a loop of herniated bowel, but this could not be verified by opaque meal or enema. Operation showed a diaphragmatic hernia, with a serous sac formed by fused peritoneum and pleura, which contained the spleen. This was replaced through the herniated orifice, which was $2\frac{1}{2}$ inches in diameter, and the patient made a good recovery. This case has been published by *Bryce and Gray*.



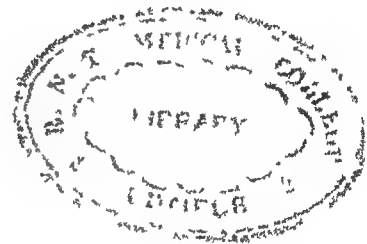
FIG. 84.—Diaphragmatic hernia of liver. The triangular shadow in the right chest is due to the liver. Diaphragm ruptured by injury. Operation confirmed. The liver lay in the pleural cavity.

(2) **HERNIA OF LIVER.**—Following a severe crushing injury, the patient a young man, was admitted acutely ill. The X-ray examination showed a triangular opacity in the lower right chest, continuous with the heart shadow. Outside this was a clear band (a pneumothorax). The shadow was of greater density than a collapsed lung of the same size. A diagnosis of diaphragmatic rupture with hernia of liver and pneumothorax was made (Fig. 84). Subsequent X-ray examination showed pneumoperitoneum to be present, as well as pneumothorax. The accidental pneumoperitoneum enabled the writer to

demonstrate absence of the liver from its normal position. At operation (Prof. Morley) the entire liver was found to be herniated into the pleural cavity through a rent in the diaphragm, to the right of the heart. The tear, about 4 inches long, ran through the tendon and muscle in a roughly antero-posterior direction. After enlargement of the opening, the liver, the vascular pedicle of which was intact, was returned to the abdomen. The patient, however, succumbed to his injuries.

TUMOURS OF THE DIAPHRAGM

When a rounded shadow is seen projecting upwards from the diaphragm, primary tumour of the diaphragm must be considered as a possibility. These are rare, very few cases having been reported. An angiosarcoma was reported by *Burrill-Holmes* and *Brodie*. It arose from the postero-superior surface and measured 12 by 6 cm. Lipoma of the diaphragm has been demonstrated by *Stewart* and *Illick*, dermoids by *Hedblom* and *Harcington*.



CHAPTER XIV

THE LUNG FIELDS

IN STUDYING a normal postero-anterior radiogram, the right and left sides are distinguished by the following points :

There are four chief landmarks on the left side and three on the right. *On the left*. (1) the prominent left heart border and apex ; (2) the prominence of the aortic arch, continued into the descending aorta ; (3) the translucent air in the fundus of the stomach, or splenic flexure of the colon, immediately below the diaphragm ; and (4) the clear-cut upper border of the left hilum (left pulmonary artery) with the left bronchus below it. *On the right side* : (1) the right diaphragm, higher than the left, with the dense liver shadow below it ; (2) the indistinct upper border of the hilum ; (3) the hair-line of the right middle interlobar pleura, sometimes visible.

Except in a case of transposition of the viscera (easily recognised on screen examination) it is almost impossible that *all* these landmarks will fail simultaneously, but when gross changes are present in one or both lungs, the landmarks may be reduced to one or two.

THE HILAR AND PULMONARY SHADOWS

On either side of the mediastinum lies a crescentic shadow of medium density—the hilar shadow—which on its outer side breaks up into fine branches which radiate out into the lung fields and can normally be traced to within about half an inch of the chest walls. All these shadows are mainly vascular in origin. For many years they were regarded with suspicion as pathological structures. Radiologists and physicians believed that any increase or supposed increase in the distinctness with which they could be seen must necessarily be interpreted in terms of inflammatory or fibrotic peribronchial change. Even to-day, when their true nature is more generally understood, there is a tendency to read into such variations more than the facts would warrant, and the word “ fibrosis ” springs all too readily to the mind in explanation of them.

The evidence that the lung shadows are due to vessels is convincing, and may be here briefly recapitulated.

Internal Evidence of Normal Radiograms.—(1) In the left hilum the uppermost structure is the left branch of the pulmonary artery. A considerable interval exists between its upper border and the upper border of the bronchus in the radiogram, and there is no structure other than the artery which could cast this shadow. Taking this as a starting-point, it is quite easy to trace its

branches out into the lung field. Where they run end on they are seen as rounded spots. Here, at all events, simple observation shows that the pulmonary artery and its branches can account for the normal lung shadows. 2) When a lung is partially collapsed by pneumothorax, its arborisations become more slender, and almost disappear, though the lung remains fairly translucent. The inference that this is due to reduction in the calibre of the vessels, is a result of diminution in the amount of blood carried by them, is supported by observation of the opposite lung, which shows a coarser and richer network of vessels than normally, because the acting lung is now taking almost the entire blood supply from the right heart. (3) In mitral stenosis, with back pressure, and in some congenital heart lesions, the increase in the calibre of the larger pulmonary vessels and the increase in the shadows of the finer lung vessels are obvious.

Experimental Evidence.—Injections of the bronchi and of the vessels by *Garcin*, *Wasson*, and others in the cadaver, and by *Assmann* in the dog, showed that the course of the arteries, at all events in the hilar regions, was different from that of the bronchi. *Weingartner* confirmed this in the living patient by introducing opaque wires into the bronchi and showing that these lay in the inner side of the vessels. The work of *Moniz* and *Lopo de Carvalho* affords still more convincing evidence of the part played by the pulmonary arteries. By injecting sodium iodide into the right heart through a long catheter inserted through the veins of the arm in a living patient, they obtained a striking intensification of the shadows of the pulmonary arteries and their branches. This is shown in radiograms (Fig. 85) which Professor *Moniz* has contributed to this section.

The final and complete proof is given by the work of *Chaoul* and *Greineder* in their studies of the lung details by tomography.

THE PULMONARY VESSELS

The common pulmonary artery is directed backwards from the conus. It gives off the right and left pulmonary arteries.

The **Right Pulmonary Artery** passes under the aortic arch below the tracheal bifurcation and crosses in front of the right bronchus between its eparterial and hyparterial branches. It divides here into three branches, of which two go to the upper lobe; the third, descending steeply, supplies the middle and lower lobes.

IN THE **UPPER LOBE** the arterial supply takes the form of three main branches—ventral, apical, and dorsal—which accompany corresponding branches of the bronchi.

THE **MIDDLE LOBE** receives its supply by a trunk which comes off from the descending branch of the right pulmonary artery; directed forwards, it divides into a dorsal and two ventral branches.



FIG. 85.—(a) Normal lung—before angiopneumography. (b) Angiopneumogram. Catheter passed through vein of left arm into superior vena cava. Injection of sodium iodide. Pulmonary arteries and their branches strongly intensified by the opaque injection (*Egna Moniz*)

THE LOWER LOBE is supplied by the rest of the descending branch. This lies along the outer and upper side of the hyparterial bronchus and of its branches, and gives off three branches which descend, namely, ventral, middle and mediastinal, and one which ascends, namely the dorsal branch to the apex of the lower lobe. These accompany and ramify with corresponding bronchi.

The Left Pulmonary Artery passes obliquely backwards from the common stem into the lung. It is seen in the radiogram just below the aortic knob, and, as pointed out by *Delherm and Chaperon*, forms the free crescentic upper margin of the left hilar shadow, above the downwardly curving left bronchus. In thin people its shadow can be seen, on the screen, to darken with each contraction of the heart, and this pulsation can be traced outwards for a little distance into the lung field.

In the left oblique view the shadow of the artery is seen passing backwards across the clear space below the aortic arch.

Arching over the left main bronchus, it divides before it enters the hilum into several branches, usually three in number, and these rapidly subdivide into nine principal branches of supply to the left lung. Five go to the upper lobe, namely ventral, apical, and dorsal, as already described in the right upper lobe, and two to the lower anterior part of the lobe. The last mentioned can be seen in radiograms as curved shadows to the outer side of the left heart border. In the lower lobe there are four branches, ventral, middle, and mediastinal (descending), and dorsal (ascending), as in the right lower lobe.

Pulmonary Veins.—The arrangement of the pulmonary veins in the peripheral parts of the lung is different from that in the central parts. In the peripheral parenchyma the veins do not run with the arteries, but are

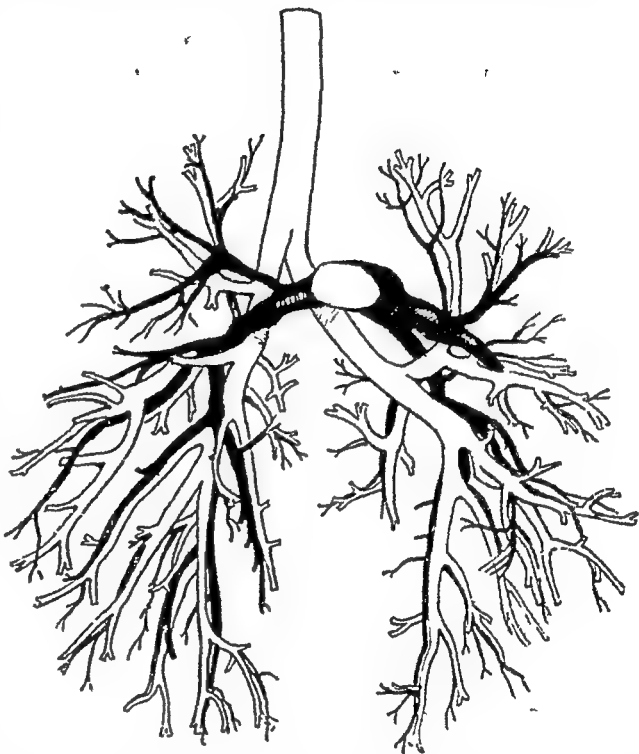


FIG 86 —Diagram : Pulmonary artery and bronchi. The branches of the pulmonary artery (black) lie for the most part above and to the outer side of the bronchi which they accompany. At the apices and extreme bases they are shown in the diagram to the inner side of the corresponding bronchi (After Narath)

situated in the septa which unite several lobules into "sublobes," whereas the arteries ramify in the lobules. Throughout the finer divisions of the lung parenchyma this separation of the veins from the arteries is maintained, as *W. S. Miller* has shown.

The veins and arteries come together at about the fourth bronchial bifurcation and are more closely related, the veins lying anterior to and below the

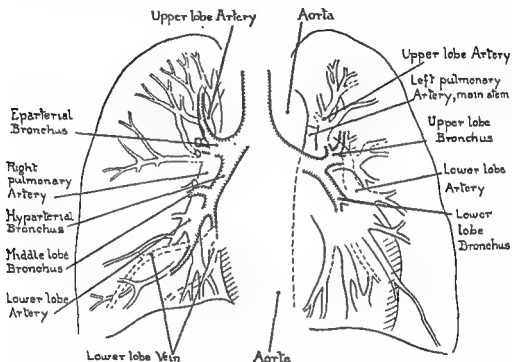


FIG. 87.—Diagram: The pulmonary arteries and veins in relation to the bronchi, as shown by tomography (After Greineder)

arteries. As they approach the hilum they again become dissociated, the veins lying below and anterior to the arteries and diverging from them to enter the left auricle. The veins are not as a rule clearly distinguishable from the arterial trunk shadows in radiograms, though the large vein from the lower lobe can sometimes be dimly made out on the right side as it enters the lower part of the hilum. It can be very clearly seen as a separate structure in this situation in tomograms of the lung in postero-anterior, lateral, and oblique views (*Chaoul, Greineder*) (Fig. 87).

CHAPTER XV

THE LOBES AND INTERLOBAR FISSURES OF THE LUNGS

THE LUNGS are divided into lobes by deep fissures, which form the surfaces of contact of the lobes. Each lobe is closely invested with visceral pleura. At the bottom of each fissure this pleura is continuous with that covering the neighbouring lobes. During breathing the lobes glide upon one another.

The fissures are of radiological importance in connection with (1) interlobar effusion; (2) interlobar sclerosis; and (3) limitation of consolidations at the interlobes.

It is important that their anatomy should be visualised, (1) in connection with the lobes, and (2) in connection with their bronchial supply. In connection with the lobes, it follows, from the description of the interlobar fissures as the contact surfaces of the lobe, that their position and shape are determined by the position and shape of the lobes themselves. In the normal there are considerable variations in the depth to which they penetrate, in their inclination and curvatures, and in their number. For the sake of clarity, they will be considered first in a schematic and simple way, secondly the normal divergences from this simple scheme, and normal variations will be considered. In textbooks of anatomy stress is laid rather upon the surface markings of the interlobar fissures, that is, the *interlobar incisuræ*, on the surface of the lung. From a radiological viewpoint, the actual shape of the surfaces (*interlobar fissures*) is of more importance.

SCHEMATIC CONSIDERATION OF THE INTERLOBAR FISSURES

If a model of the lung were made, not yet divided into lobes, and viewed from the external aspect, the fissures could be imitated roughly in the following manner:

While the observer views the outer side of the model of one lung, let a knife be laid upon the dorsal surface some 3 inches below the apex, and an oblique cut be made downwards and forwards to the lower anterior margin: this would represent the oblique fissure. The incision would go right through to the mediastinal surface above the lung root, but as the hilum region was approached, the knife would be gradually withdrawn and its point would not cut into the root of the lung. Below the hilum it would again penetrate to the mediastinal surface. The effect of this slanting cut would be to divide the lung into an antero-superior half, and a postero-inferior half, held together by the hilum and a bridge of undivided parenchyma in the hilar region. This would represent roughly the condition present in the left lung. In the right lung an

additional cut would have to be made to represent the horizontal fissure. Entering the knife horizontally about half-way down the oblique fissure on the outer side, until its point reached the bottom of the previous incision, the edge of the knife would be turned forwards and a nearly horizontal incision made forwards to the front surface of the lung. The point of the knife would not now penetrate the mediastinal surface of the lung, except at its extreme anterior margin. This incision would divide the middle lobe from the superior, leaving it connected to the upper and lower lobes by hilar structures and parenchymatous bridges. To complete the model, these fissures would be lined with a "pleural membrane" closely investing the lobes thus demarcated, and continuous with the peripulmonal pleural investment of each lobe, allowing the

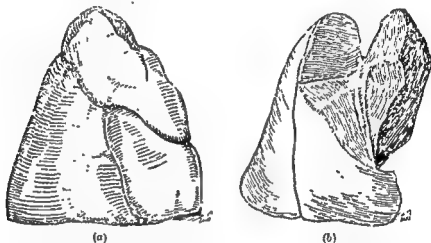


FIG. 88.—Diagram: (a) The incisura of the right lung, outer surface. (b) Upper lobe turned forwards, showing upper part of main fissure, and middle fissure. (After Kreuzfuchs and Schumacher.)

surfaces of contact of adjacent lobes to glide freely over one another, and to separate easily.

This simple scheme of the interlobar fissures fails to convey the entire truth, and they must be studied in more detail. Kreuzfuchs and Schumacher have made a careful study of the interlobar surfaces in forty autopsy specimens in children, and have described the normal shape of these surfaces, and the variations in the depth of the incisura which may be met with. Fleischner has carried the work farther in a study of the variations in position which occur in disease and the appearances produced by pleuritic processes affecting the fissures.

Shape of the Interlobar Surfaces

Main Fissure.—On the left side the surface has a crescentic shape. On the right side it is an elongated semi-ellipse (Fig. 90 (e), (f)).

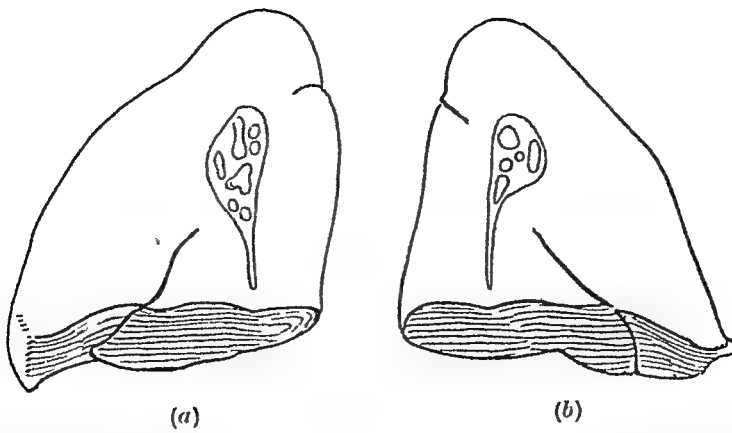


FIG. 89.—Diagram : Mediastinal surface of right lung (a) and left lung (b). The main fissure reaches to a variable distance from the hilum. The middle fissure is closed on the mediastinal surface.

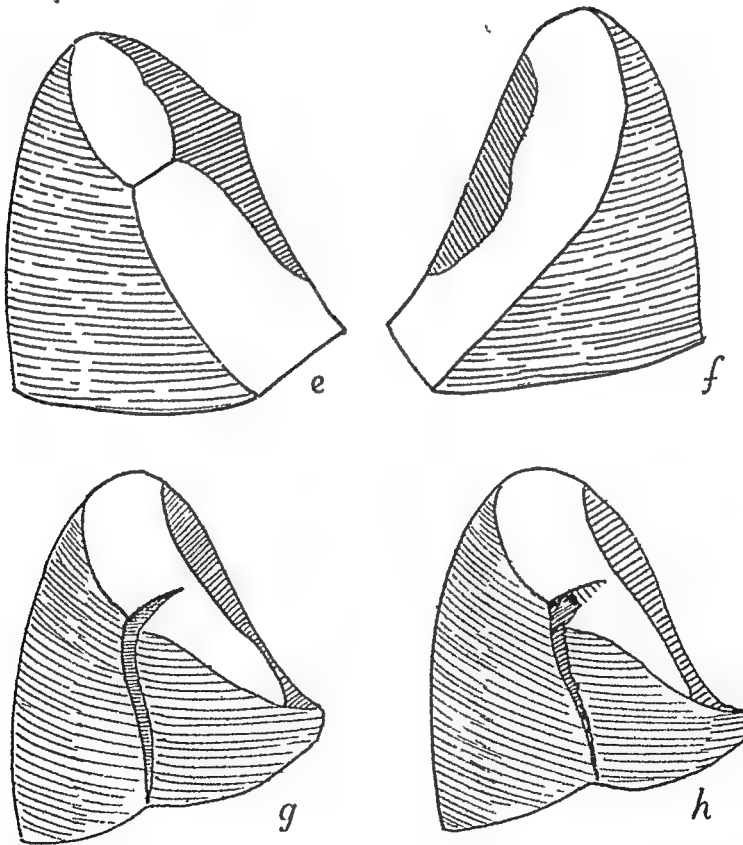


FIG. 90.—Diagram : Interlobar surfaces. (e, f) The interlobar surface of lower lobes, right and left : the parenchymatous bridges uniting them to the upper lobes (shaded). (g, h) Right lung. Middle lobe in situ : in h, a pleural bridge unites lower lobe to middle lobe, forming a continuous surface, over which an interlobar effusion may track. (After *Kreuzfuchs* and *Schumacher*.)

The Middle Interlobar Space.—This is triangular with its apex directed forwards. The inner side of the triangle (the line of reflection of interlobar pleura from middle to upper lobe) runs almost directly antero-posteriorly. The base of the triangle is formed by the junction of the middle fissure with the oblique fissure (Fig. 90 (g)).

Variations in Depths of the Fissures

The depth of the fissures is very variable. In many cases (50 per cent., Schall and Hoffmann) the main fissure fails to reach the mediastinal surface,

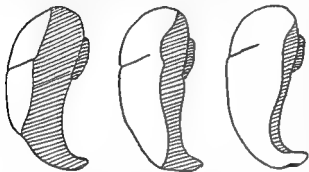


FIG. 91.—Diagram Views of right lungs after removal of upper lobes. Shaded, cut surface of parenchyma uniting upper lobe to lower and middle lobes. These bridges may be massive or narrow, and the fissures correspondingly shallow or deep (Kreuzsachs and Schumacher.)

above or below the hilum. This fissure may thus be closed on this surface by pleura, or by parenchymatous bridges of greater or less thickness (Fig. 91). This closure on the mediastinal surface occurs in about two-thirds of the cases (upper part) and in five-sixths of the cases (lower part). Even on the diaphragmatic surface closure may occur towards the mediastinal surface by a parenchymatous bridge. The middle fissure rarely reaches the mediastinal surface, and then

only at its anterior end. The middle fissure and the main fissure may both be shallow, so that a continuous surface is formed by which an effusion starting in the upper part of the main fissure is encouraged to track downwards on to the upper surface of the middle lobe. The same thing may occur if there is a bridge of pleura from upper surface of middle lobe to upper surface of lower lobe (Fig. 90 (h)). If the middle fissure is shallow, an effusion in it will be found in the outer part of the lung field. If deep, the effusion may reach inwards nearly to the hilum. Owing to the closure of the middle fissure on its mediastinal side, a mediastinal effusion does not penetrate into it, whereas it frequently happens that a mediastinal effusion enters the main fissure (mediastino-interlobar effusion).

Projection of the Incisurae on the Surfaces of the Lung

If the incisura of the oblique fissure is followed from its mediastinal surface above the hilum over the outer surface of the lung to its end on the mediastinal surface below the hilum, it will be found that it rises steeply on the mediastinal surface and falls more obliquely on the outer side. On gaining the lower

anterior margin of the lung, it turns backwards along the diaphragmatic surface to gain the mediastinal surface, where it runs nearly vertically towards the hilum. As a result, the upper part of the interlobar surface of the lower lobe looks forwards and *outwards*, while the lower part of the surface looks forwards and *inwards*. Viewed from the side, these surfaces are twisted relatively to one another like the blades of a propeller (*Diellen*).

The upper limit of the lower lobe on the posterior surface of the lung is at the vertebral end of the third rib (medial end of spine of scapula). The main interlobe cuts the inferior margin opposite the lateral part of the sixth costal cartilage (*Cunningham*).

In the right lung the horizontal incisura starts from the main incisura where the latter crosses the posterior axillary line and ends anteriorly at the level of the fourth costal cartilage.

The incisura reaches the lower anterior margin of the right lung rather more than a

hand's breadth from its anterior margin. In the left lung this point is nearer to the anterior margin, owing to the presence of the cardiac notch. About one-eighth of the diaphragmatic surface of the left lung is formed in its antero-median part by a very small portion of the upper lobe. On the right side a rather larger part of the middle lobe is in contact with the diaphragm. This may account for as much as one-half of the diaphragmatic surface of the right lung, but is usually much less in normal chests. When the right

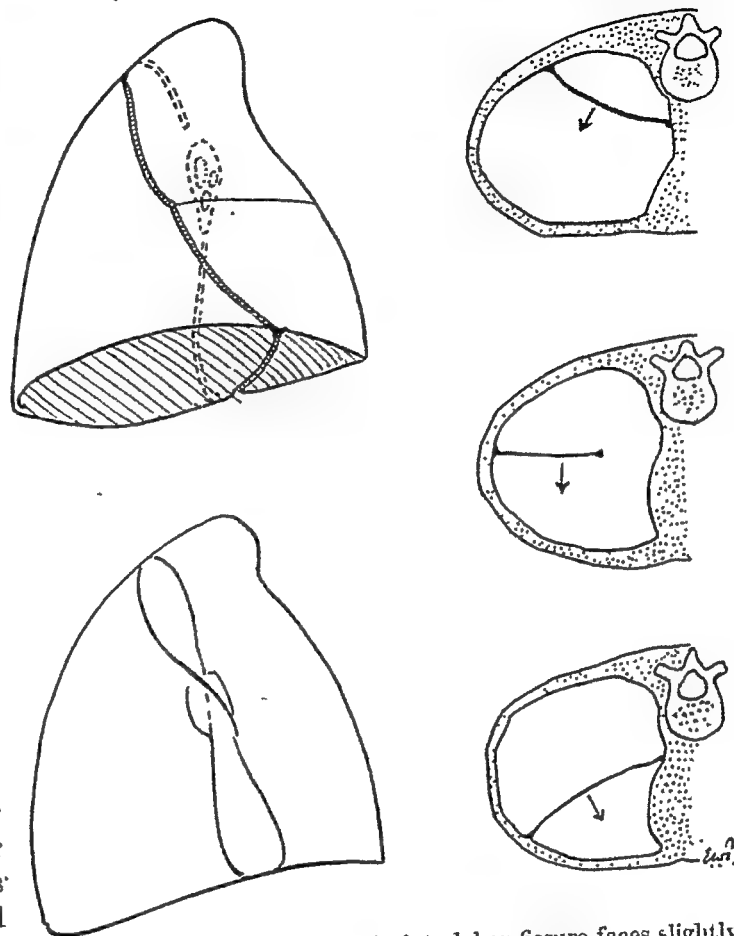


FIG. 92.—Diagram: The main interlobar fissure faces slightly outwards in the upper part of the chest, slightly inwards in the lower part, as shown in the series of cross sections (right). Its projection on the outer surface of the lung is therefore propeller-shaped. (*Diellen*.) Slight rotation of the patient may therefore be required to show it edge-on in the lateral view.

lobe is diminished in volume, the middle lobe is displaced upwards and forwards, and the area of its diaphragmatic surface is correspondingly reduced. If the middle lobe is atelectatic, it usually ceases to have any contact with the diaphragm, and the lateral view shows it to have the form of a triangle, the base of which abuts on the anterior chest wall. If the *lower* lobe is atelectatic, on the other hand, the middle lobe is displaced backwards and the area of its diaphragmatic surface is increased. It is therefore a cause of error to expect to find the interlobar fissures in definitely fixed positions, or to map out the lobes with reference to surface landmarks only.

Viewed from the side, the main incisura of the right lung is not straight, but forms an angle, apex forwards, at the junction with the middle incisura. Its upper part is more horizontal than on the left side. The left incisura is usually straight, but may show a similar angulation. The principal fissure is steeper on the left side than on the right, making an angle of 60 degrees with the horizontal on the left side and about 50 degrees on the right.

Curvature of the Interlobar Surfaces

THE MAIN FISSURE.—In addition to the twist in the main fissure previously noted (propeller form) there is a curvature from side to side. This curve is convex upwards, imitating the curve of the diaphragm so that the lower lobe bulges into a concavity in the upper (L) or middle lobe (R). This is certainly true as regards the lower part of the fissure, and is readily observed in the hollow-back projection. The upper part is so difficult to observe that no definite statement can be made on this point. In lungs, hardened in situ, *Kreuzfuchs* and *Schumacher* found that this was variable, and that in many cases the upper half was concave upwards, the upper lobe bulging into the lower lobe. Seen from the side, both parts of the main fissure are concave upwards.

MIDDLE FISSURE.—This is convex upwards from side to side. Seen from the side, it usually shows a double curve (S curve). Traced forwards from the hilum level, it is first convex upwards, then, near the anterior margin, concave upwards. At this point the upper lobe overlaps it. The general direction is horizontal, but in many cases in life there is a pronounced slope forwards and downwards.

The double curvature may cause a duplication of the hair-line of the middle fissure in postero-anterior radiograms, if the ray becomes tangential to both portions of the curve.

RADIOLOGY OF THE INTERLOBAR FISSURES

Normal interlobar pleura, although thin, becomes directly visible in a radiogram if any considerable extent is struck tangentially by the rays. Radiologists are familiar with the appearance of the horizontal fissure, so often seen in postero-anterior radiograms. Its frequent appearance in this projection is

readily understandable from Fig. 93. A hair-line in this situation may be seen in normal chests and has been found in new-born children. The conclusion of *Hotz*, that its occurrence in children is evidence of tuberculous infection, cannot be supported.

The interlobar pleura of the main fissure is not visible in the postero-anterior view because it runs across the rays and not tangentially. Illumination along the interlobar fissure by raising the tube or by the adoption of the hollow-back position will often render it visible. The lateral position often brings both

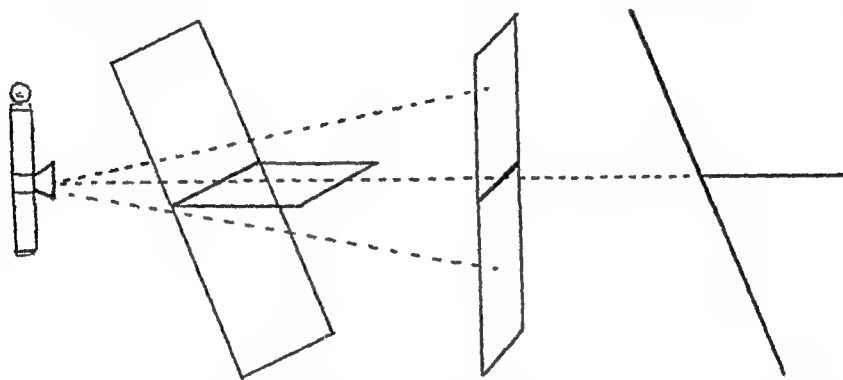


FIG. 93.—Diagram: The interlobar fissures illustrated schematically by cards. In the P.A. view the middle fissure, edge-on to the rays, casts a fine shadow. The main fissure, face-on to the rays, is invisible. In the lateral view both fissures, edge-on, are visible.

fissures into prominence, because in this position there is a high probability of a considerable extent of interlobar pleura running in the line of the rays. It must, however, be remembered that the oblique fissures do not run in an exact coronal plane, and that the main fissure is normally twisted propeller-wise; also that the middle fissure may slope downwards and outwards or upwards and outwards. Suitable degrees of rotation of the patient, or of inclination of the patient's body towards or away from the screen or film, may, therefore, be necessary for best visualisation. Sometimes the twisting of a fissure may be visible, the twisted planes of interlobar pleura at different depths giving an appearance described by *Eisler* as the "banner sign" from its resemblance to a pennant twisted by the wind.

Whenever disease is present in the lung, the interlobar pleura is liable to be visibly thickened because it is richly supplied with lymphatic vessels, and these constitute the principal drainage channels from the peripulmonary pleura towards the hilum. The nearer parenchymatous lesions are to an interlobar surface, the greater the frequency of interlobar thickening. It is therefore found in most inflammatory conditions of the lung, especially in pneumonia, and often in lung abscess, tuberculosis, and malignant disease. It may also be observed in the "back-pressure" lung of mitral stenosis in lateral films.

The radiological demonstration of the interlobar fissures enables the radio-

logist to localise a gross lesion in its correct lobe, or part of a lobe, with great precision, but it is essential that the lateral view be used to supplement the postero-anterior, and moreover that the radiologist should not rely upon what *Fleischner* has called "blind radiography," but should precede radiography by very careful fluoroscopy at ever-varying angles, until a most suitable position is found.

Observations of the Interlobar Fissures in Atelectasis and Fibrosis.—Displacements of the interlobar fissures in atelectasis of a lobe, or fibrosis, may alter the normal relationships to a remarkable extent. It is common knowledge that compensatory emphysema of the unaffected lobe rapidly occurs and that the appearance in the postero-anterior view may be very little altered. We may, for example, see a not very large triangular opacity at one apex, or a triangular opacity bordering the heart at one base, with an otherwise normally

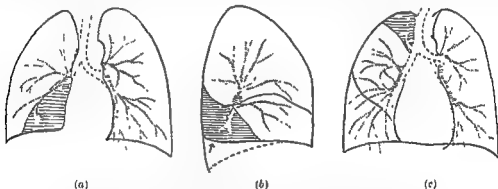


FIG 91.—Diagram Identification of the vascular shadows as an aid to the study of lobar collapse and fibrosis. (a) and (b) Lower lobe collapse. The vascular shadows of the lower lobe have disappeared. The remainder have spread out fanwise. The hilum is displaced backwards and the diaphragm is elevated posteriorly (b). (c) Upper lobe collapse or fibrosis. Upper lobe vessels invisible. Middle lobe vessels displaced upwards.

translucent hemithorax on this side. The beginner does not readily associate with such appearances the true explanation, namely total atelectasis of the upper or the lower lobe respectively. Yet he can easily convince himself that this is the true explanation by observing the position of the interlobar fissures. In the case of right upper lobe collapse, the horizontal fissure, pivoting on the hilum, has swung from a horizontal into a steeply ascending position. In the case of the lower lobe collapse, the lateral view shows the oblique fissure to be displaced downwards and backwards. In the case of a mid-lobe collapse, the middle lobe in the lateral view occupies a narrow triangle in the lower anterior part of the chest. The emphysematous upper lobe bulges downwards over it, the lower lobe bulges forwards below it.

If observation of the vascular trunk shadows is combined with this observation of the position of the interlobar fissures, a still clearer conception of the

atelectasis and compensatory emphysema is obtained. The trunk shadows must for this purpose be traced back to their points of emergence from the hilum, from which they radiate. These "radiant points," as the writer has elsewhere described them, are constant for each lobe, and can be memorised when the anatomy of the bronchi has been learned, for the vessel shadows run with the bronchi through the lung parenchyma. It will be found that the trunk shadows of the atelectatic lobe are lost in the obscurity of this lobe, while the trunk shadows visible in the translucent part of the lung can be identified as belonging to the other lobes (Fig. 94). If the atelectasis is a partial one, affecting only a wedge-shaped area of one lobe, the trunk shadows of the unaffected portion of that lobe can still be identified.

ACCESSORY LOBES OF THE LUNGS

Retrocardiac (Inferior Accessory) Lobe

The territory supplied by the retrocardiac bronchus on the right side, or by the corresponding branch of the anterior basal bronchus on the left side, may

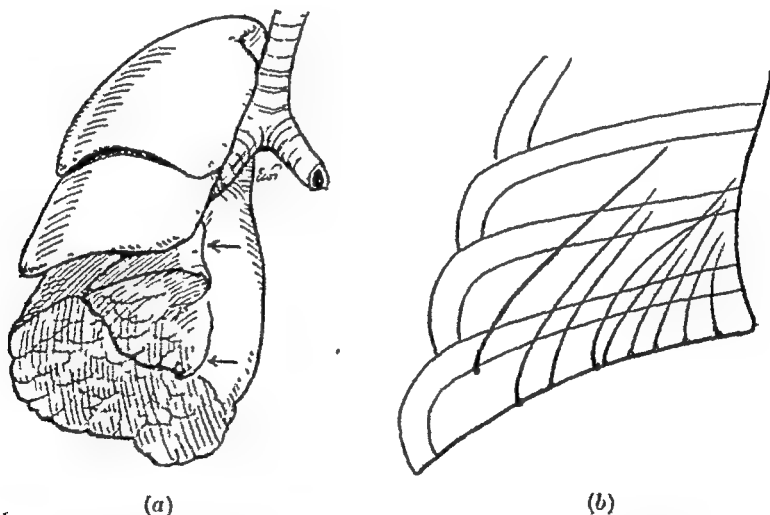


FIG. 95.—Diagram: (a) Retrocardiac (Infiacardiac) accessory lobe, (b) Different positions in which the fissure of the retrocardiac lobe may be seen

exist as a separate lobe in man, as it does in nearly all quadrupeds. The right retrocardiac bronchus, originating about 1 cm. below the posterior horizontal bronchus, aerates the posterior two-thirds of the inner aspect of the lower lobe (*Ewart*). A somewhat smaller area is aerated by the corresponding bronchus on the left side.

As a result of X-ray investigations *Kerley* has demonstrated the right retrocardiac accessory lobe in the chimpanzee. In this ape it is constantly present. *Gräberger* has drawn attention to the radiological appearances which may be

due to this lobe in health and disease. It is of interest (a) because its interlobar fissure may be visible, (b) because it gives rise, when atelectatic or consolidated, to a triangular shadow which has to be differentiated from other conditions, and (c) because it is likely to be the seat of a bronchiectasis or congenital cystic condition.

This lobe was called by *Owen* the *Lobus Impar*, and by *Broca* *Lobus Azygos*. The latter term is inconvenient, since it may give rise to confusion with the lobe of the azygos vein, now universally known to radiologists as the "azygos lobe." Other names by which it is known to anatomists are cardiac or infracardiac (*Aeby*), retrocardiac (*Ewart*), inferior accessory lobe (*Reztorzik*). This name, inferior accessory lobe, which it received from *Reztorzik*, who first described it in the human in 1861, is distinctive enough for all practical purposes.

ANATOMY.—The accessory lobe represents a wedge-shaped volume, of varying size, anterior to the pulmonary ligament, with its base on the diaphragm, and its apex at the lower part of the lung root. Its median surface forms part of the mediastinal surface of the lung, below the root. Its interlobar surface slopes upwards and inwards to the root. It is sometimes completely developed, with a tongue-shaped anterior extremity; in other cases the division may be partial, or even merely indicated on the surface by shallow fissures or indentations.

FREQUENCY.—This lobe has been found in 43 per cent. of 210 post-mortem lungs, either fully or partly developed (*Schaffner*). Radiologically it has been found in 8 per cent. of 500 unselected radiograms. In two cases an azygos lobe was present as well. *Richards* states that he found it only ten times in 2,000 consecutive radiograms.

RADIOLOGICAL APPEARANCES.—*Assmann* described in his book a case as follows: "I saw in one case a basal triangular paravertebral shadow (apparently due to an infiltrated inferior accessory lobe) on the left side seen through the heart shadow, proved by rotation of the patient to be behind the heart, and therefore exactly resembling a left-sided costo-mediastinal pleurisy. Autopsy, however, revealed an infiltration confined to an abnormal third left lobe."

Ettig and *Schonfeld* described a chronic pneumonia in a left-sided inferior accessory lobe.

Gräberger observed a case of pneumonic infiltration in this lobe which cleared up in a few weeks, leaving only a faint interlobar line extending downwards to the diaphragm from the hilar region.

This accessory interlobe will frequently be seen in radiograms if carefully looked for. It sometimes ends at the diaphragm in a triangular peak, similar to those to which the writer of this section has drawn attention at the lower end of the main interlobe; but the direction of the line is different in the two cases—namely upwards and inwards in the case of accessory fissure, upwards and outwards if due to the main fissure.

DIFFERENTIAL DIAGNOSIS—As regards the difficult differential diagnosis

between an infiltrated accessory lobe and an anterior or posterior costo-mediastinal exudate, *Gräberger* believes that—

(a) The anterior encapsulated effusion will show a convex outer border.

(b) The inferior accessory lobe shows a straight border.

(c) The posterior costo-mediastinal pleural thickening will show a slightly concave border.

In the case of (b), however, if atelectasis is present, a concave border will occur.

Fleischner has also drawn attention to the difficulty in making the distinction between a posterior costo-mediastinal thickening and consolidated accessory lobe. The movements on respiration may decide the point. The edge of a costo-mediastinal pleurisy will preserve a constant relationship to the chest wall, while that of the accessory lobe will tend to move towards the midline on inspiration, being displaced by the inflation of the remainder of the lower lobe. *Fleischner* has also noted the variation in the direction of the fissure of this accessory lobe, in different degrees of development, and the need for suitable oblique illumination in order to show it.

Atelectasis of the lower lobe may be hard to distinguish from an accessory lobe. The observation of the bronchial trunks may give a clue. If the lower lobe trunks can be clearly identified, passing downwards to the outer side of the shadow, the shadow must be due to an accessory lobe (or a pleural condition). In some cases only the use of lipiodol will enable one to decide whether the whole lower lobe bronchial supply or merely the retrocardiac bronchus is included in the shadow.

A basal triangular shadow should be carefully scrutinised for possible presence of bronchiectatic cavities within it. If atelectatic and containing bronchiectatic cavities, the case may be one of primary congenital atelectatic bronchiectasis. Lipiodol should be employed in order to make sure of the presence or absence of bronchiectasis in such a case, and to decide whether a fourth lobe or an entire lower lobe is involved.

In the writer's opinion the majority of basal triangular shadows are due to acquired atelectasis of a lower lobe, a condition which is not uncommon in children, in whom the entire lower lobe or lobes may rapidly collapse to very small dimensions. Bronchiectasis can develop rapidly in such a collapsed lobe as a result of subsequent infection.

Richards has demonstrated bronchiectasis in this accessory lobe. Other cases are described by *Kerley*, who has also seen a case of tuberculous infiltration in it. Cases of malignant involvement of this lobe are also recorded.

In the differential diagnosis must also be considered :

(1) Enlargement of left ventricle.

(2) Cold abscess of spine.

(3) Para-oesophageal hernia.

The line of the accessory interlobe may be simulated by that of a para-

oesophageal hernia, in which case the line is due to the stomach wall, is thicker and usually convex outwards, with a translucent air shadow to the medial side in the right costophrenic angle.

The hernia is by no means obvious on the usual chest radiogram. Barium meal clears up the diagnosis.

(4) An abnormally placed inferior vena cava filling the right cardiophrenic angle.

(5) Abnormal pericardial fat. It has been shown by *Kautz and Pinner* that large extrapericardial fatty masses may occur of sufficient size to simulate a small accessory lobe. With careful screening, however, the diagnosis should not be difficult.

Posterior Accessory Lobe of the Lung

Pohl states that this lobe, which occupies the apex of the right lower lobe, is not a very rare anatomical variation. It corresponds with the distribution of the first dorsal bronchus, and originates as a result of non-fusion of the right dorsal apical bud of the *anlage* of the lower lobe bronchus with the rest of the lower lobe. This bud is normally present from the fifth foetal week on both sides, but fuses later with the remainder of the lower lobe (*Keibel and Mall*).

ANATOMY.—The horizontal cleft between upper and middle lobes is continued backwards, sometimes without interruption, at other times leaving a band of lung tissue, usually on the mediastinal side, which connects the accessory lobe with the lower lobe. Rudimentary forms occur in which there is only a superficial indication of an accessory cleft.

RADIOLOGICAL APPEARANCES—If this lobe is infiltrated with pneumonia, a triangular dense area is seen in the lateral view, sharply limited on both sides, fading off in density towards the chest wall, which may be mistaken for an interlobar effusion, which in this situation is not necessarily spindle-shaped. The writer has seen an effusion occupying the posterior upper end of the main interlobe, partly parietal and partly interlobar, which was wedge-shaped in the lateral view. In this case the diagnosis was given by its bulging upper and lower contours. It was confirmed by operation.

A consolidation of this region of the lung, or an interlobar effusion above or below the accessory lobe, shows a shadow in the postero-anterior view at about the fifth or sixth posterior interspace, close to and often blending with the hilum. This is a favourite area for the development of pneumonia in childhood: many of the "central" or "hilar" pneumonias (so described) are actually in this territory of the lung, but have been incorrectly localised because no lateral view was taken.

Leviten and Brunn believe that this accessory lobe is of frequent occurrence, basing their opinion upon the frequency with which this sector of the lung may show isolated consolidation, or escape when the rest of the lobe is consolidated. The writer agrees as to the frequency of such isolated involve-

ment, especially by abscess, but believes that this does not necessarily signify the presence of an accessory lobe. It is caused rather by limitation of the disease to the region supplied by the first dorsal bronchus, an area which is separated from the rest of the lobe by an "avascular" zone (see Fig. 164). The writer has looked closely for evidence of sclerosis in an interlobar fissure here for the last ten years, since becoming aware of the existence of this separate lobe, but has found this interlobar fissure to be exceedingly rare, and has only observed it on two or three occasions. It is not necessary to assume the presence of an accessory lobe whenever an isolated sector of lung is dense, since this can be amply explained by the bronchial distribution; for other segments of the lining, in which there is no known accessory lobe, may likewise present similar triangular densities when atelectatic or consolidated.

Middle Lobe of the Left Lung

The region supplied by the ventral ("middle") branch of the left upper lobe bronchus is the homologue of the right middle lobe, and is in fact demarcated from the rest of the lobe by a relatively avascular zone. A fissure sometimes occurs which separates it off as a left middle lobe; this fissure is usually incomplete, and merely indicated on the surface by a slight furrow. A case has been described in the anatomical literature by Maylard. No instance of radiological recognition of this accessory lobe is known to the writer.

Accessory Azygos Lobe

DEVELOPMENT.—In early embryonic life the azygos vein runs over the right apex, but as the latter grows upwards, the vein is pulled down by the

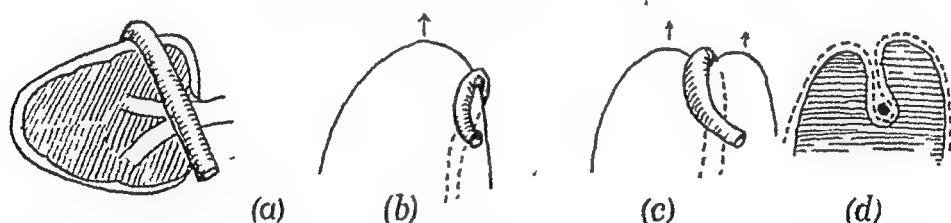


FIG. 96.—Diagram: Formation of the accessory azygos lobe. (Lobe of the azygos vein.) (a) and (b) The vein normally becomes displaced to the inner side of the growing lung. (c) and (d) If this displacement is arrested, the vein becomes embedded in the growing lung, carrying with it a fold of parietal pleura.

heart on to the medial side of the apex and finally arches over the lung root. If this gliding movement is arrested, the venous loop of the azygos vein indents the lung. As the lung grows, the vein, carrying with it a fold of pleura (parietal and visceral), becomes deeply embedded in it. An accessory lobe of varying size is thus separated from the medial aspect of the apex. Thus the fissure of the azygos vein originally consists of four layers of pleura. It communicates

with the general pleural cavity, and a pleural effusion has been observed by the writer, entering the fissure and so broadening its originally fine line.

RADIOLOGICAL APPEARANCES.—The fissure of the lobe is seen in radiograms as a curved hair-line, always at the right apex, convex outwards, and variable in situation. At its upper end a small triangular peak is often seen. At its lower end it ends in a pear-shaped shadow above the hilum, which is due to the shadow of the azygos vein embedded in the cleft. This pear-shaped rather dense shadow may be continued downwards towards the upper border of the hilum by a fainter linear shadow, lying posteriorly due to the ascending part of the azygos vein. Sometimes the pear- or comma-shaped shadow is obvious, but the fine interlobar line is almost invisible. In these cases of

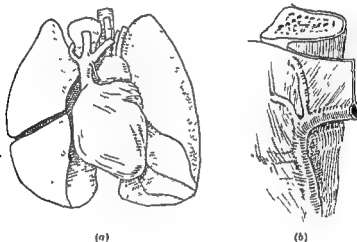


FIG 97.—Diagram. (a) Accessory azygos lobe, lying to the medial side of the azygos vein. (b) Azygos vein running forwards from the lateral aspect of the dorsal vertebra enclosed in fold of pleura (mesoazygos) (After Anson and Smith)

small azygos lobe careful scrutiny may reveal the interlobar line lying close to the shadow of the superior vena cava.

It is doubtful whether the thickening at the lower end of the fissure of the azygos lobe is entirely due to the azygos vein seen "end-on," though this is the explanation which has been generally accepted up to the present. In oblique and in stereoscopic films this part of the shadow has no resemblance to an end-on vessel, but appears to be due rather to several folds of redundant pleura which converge towards the lower end of the fine line of the fissure like "the neck of a purse when the string is tied" (*Bönniger*).

Consolidation may occur in an azygos lobe, or this lobe may escape when the rest of the upper lobe is consolidated. Tuberculosis may also be confined to it or it may escape in miliary tuberculosis. Compression of the bronchus supplying it may result from the pressure of the azygos vein, and as a result

atelectasis or bronchiectasis may occur. The fissure of the lobe is sometimes thickened by interlobar sclerosis.

CONGENITAL ABSENCE OF ONE LUNG

Patients may live to old age in spite of non-development of one lung. The condition is extremely rare. Of a series of twenty-two cases collected by

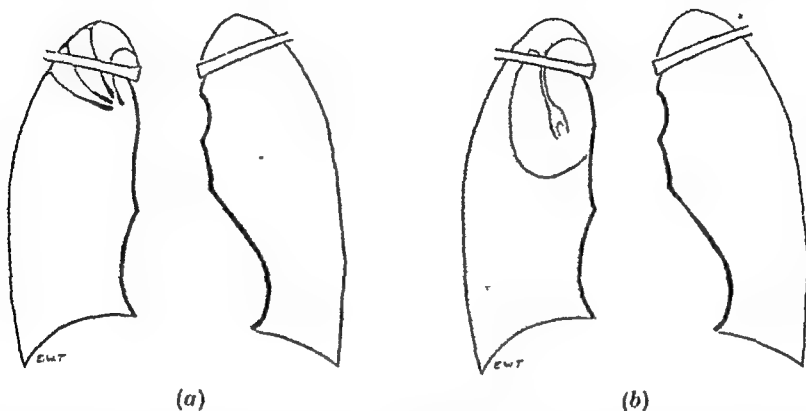


FIG. 98.—Diagram: Fissure of the accessory azygos lobe. (a) Various positions in which the fissure may be found. (b) Effusion entering the fissure.

Levy fifteen were on the left side. Duration of life was from 8 to 70 years. Respiratory symptoms were uncommon. *Elward* has recently reported a case with X-ray and post-mortem findings; the X-ray appearances were those of a unilateral fibrosis with compensatory emphysema of the opposite lung. At post-mortem the rudimentary left lung had a volume of only $4\frac{1}{2}$ cubic inches. The bronchus extended into the lung for a short distance, and ended abruptly in a series of small sclerotic and partially occluded terminal branches. *Elward* emphasises a symmetrical thorax as a differential sign, and states that the intercostal spaces are not narrowed as in acquired atelectasis or fibrosis. This is not borne out by his published radiogram, which clearly shows increased obliquity of the ribs on the affected side. A consideration of the specimen shown in Fig. 146 of a case of extreme fibrosis of one lung, with great compensatory emphysema of the other, will make clear the difficulty of making a differential diagnosis.

CHAPTER XVI

ANATOMY OF THE BRONCHI

THE DIVISION of the trachea into two main bronchi is repeated at each subsequent bronchial division, though in a few places this exact dichotomous arrangement is masked by the closeness of the repeated divisions and the unequal size of the resulting branches, so that the bronchus appears to divide suddenly at some point into three or more divisions as, for example, in the case of the main bronchus to the right upper lobe. The branches are rarely symmetrical as to size of the resulting bronchi. Nearly always one of the pair is larger than the other. This asymmetry has raised difficulties for anatomists, for it is not easy to determine whether the larger divisions, which may lie nearly in a straight line, represent a continuous stem bronchus or not. The result of a difference of opinion on this point has been that the bronchial tree is differently described in Continental and English literature. The former follows *Aeby*, who described the branching as monopodial; for example, in the lower lobe he described a single stem giving off alternate ventral and dorsal branches, following the arrangement found in quadrupeds, while English writers (*Nelson*) adopt a nomenclature based generally upon *Ewart's* classical work. *Ewart* described the branching as dichotomous and assailed the previously held theories of *Aeby*. The discrepancy is of more interest to anatomists than to radiologists. *Ewart's* nomenclature, with some modifications, is adhered to here.

The branchings and distribution of the bronchi have been worked out in great detail by *Ewart*. On this classical work, and on a study of radiograms of lipiodol injections, the following account is based. Although it is not necessary for the radiologist to study the ultimate ramifications in detail, it is essential that he should know the main branches, their main subdivisions, and the distribution of these, firstly, because a study of lipiodol films is impossible without that knowledge, and secondly, because many gross conditions in the chest, abscess, pneumonitis, atelectasis (due to foreign body or other cause), and tuberculosis, may be limited to the distribution of a bronchus or one of its subdivisions. Accurate localisation of such lesions demands a knowledge of bronchial distribution, and is necessary for the correct treatment of abscess by surgery and postural drainage. Thirdly, because of the assistance which close observation of the lung detail—i.e. of the vascular shadows which run with the bronchi—will give in localising a lesion to its correct lobe, and correct part of a lobe. The normal relationship of the lobes and interlobar fissures and bronchi may be so greatly upset by atelectasis and compensatory emphysema that the value of a study of the trunk shadows—i.e. of the vascular shadows which

accompany the bronchi throughout the lung parenchyma—cannot be overestimated. The writer has referred to this in a previous work, and it will be again dealt with under the appropriate sections.

Anatomists state that the trachea divides opposite the fourth dorsal vertebra. In radiograms the carina is commonly seen rather lower than this. The radiographic and cadaveric positions do not agree, because of the extensibility of the tracheobronchial stem. The usual position of the carina in radiograms is opposite the vertebral end of the seventh rib, but sometimes as high as the sixth or as low as the eighth.

BRONCHI OF THE RIGHT LUNG

The Right Main Bronchus, shorter than the left, and more directly in line with it, is directed downwards and outwards, and divides into two main branches :

- (1) The upper lobe bronchus (eparterial).
- (2) The continuation of the main stem (bronchus intermedius, *Ewart*; hyparterial bronchus, *Aeby*).

Right Upper Lobe Bronchus.—Arising from the outer wall of the main bronchus opposite the bifurcation of the trachea, it is directed almost horizontally outwards. It supplies the whole upper lobe. It has three branches (the trifurcation is apparent, not real, consisting of two asymmetrical bifurcations rapidly following each other).

(a) **APICAL.**—Directed upwards, outwards, and a little backwards. Divides into anterior and posterior branches. The anterior supplies the supraclavicular apex in its anterior half. The posterior supplies the posterior half of the supraclavicular apex and the inner posterior part of the upper lobe.

(b) **AXILLARY.**—Directed outwards. Divides into : (1) anterior axillary, directed forwards and upwards ; (2) superior axillary, directed backwards. These supply a pyramidal region, the base of the pyramid being on the axillary surface of the upper lobe, extending for a short distance on to the posterior part of the outer surface. The interlobar surface in this region receives a special branch from the main axillary stem.

(c) **PECTORAL.**—Directed forwards (often seen in radiograms as a circle with clear centre in the upper hilum). Divides into two : (1) the sterno-pectoral, directed inwards ; (2) mid-pectoral, directed outwards. These branches eventually supply a pyramidal region, having its base on the anterior aspect of the chest between the clavicle and the mamma, its apex at the hilum ; bounded below by the horizontal fissure.

Right Lower Main Stem.—The main stem courses downwards with a slight curvature outwards, about an inch from the right heart border, and rapidly subdivides. The first division occurs about $1\frac{1}{2}$ inches below the eparterial bronchus : at this point the middle lobe bronchus (anterior), and, close to

the posterior ("first dorsal") branch to the apex of the lower lobe are given off. Below this point the remainder of the lower lobe bronchi arise in succession.

Right Middle Lobe Bronchus (cardiac bronchus, *Ewart*).—This is given off immediately below the lower division of the right pulmonary artery, which

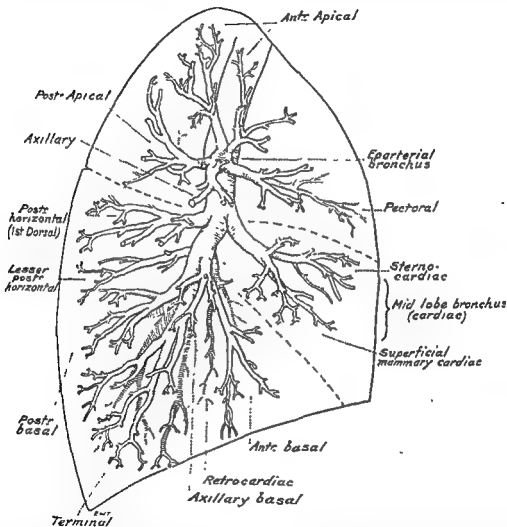


FIG. 99.—Diagram: Bronchi of the right lung: lateral view.

here crosses the main bronchus. It turns forward under the lower border of this artery, and divides into branches for the supply of the middle lobe, viz.:

- (1) INNER (STERNOCARDIAC).
- (2) OUTER (SUPERFICIAL MAMMARY CARDIAC).

The inner immediately subdivide again into inner and outer branches. A trifurcation is thus stimulated.

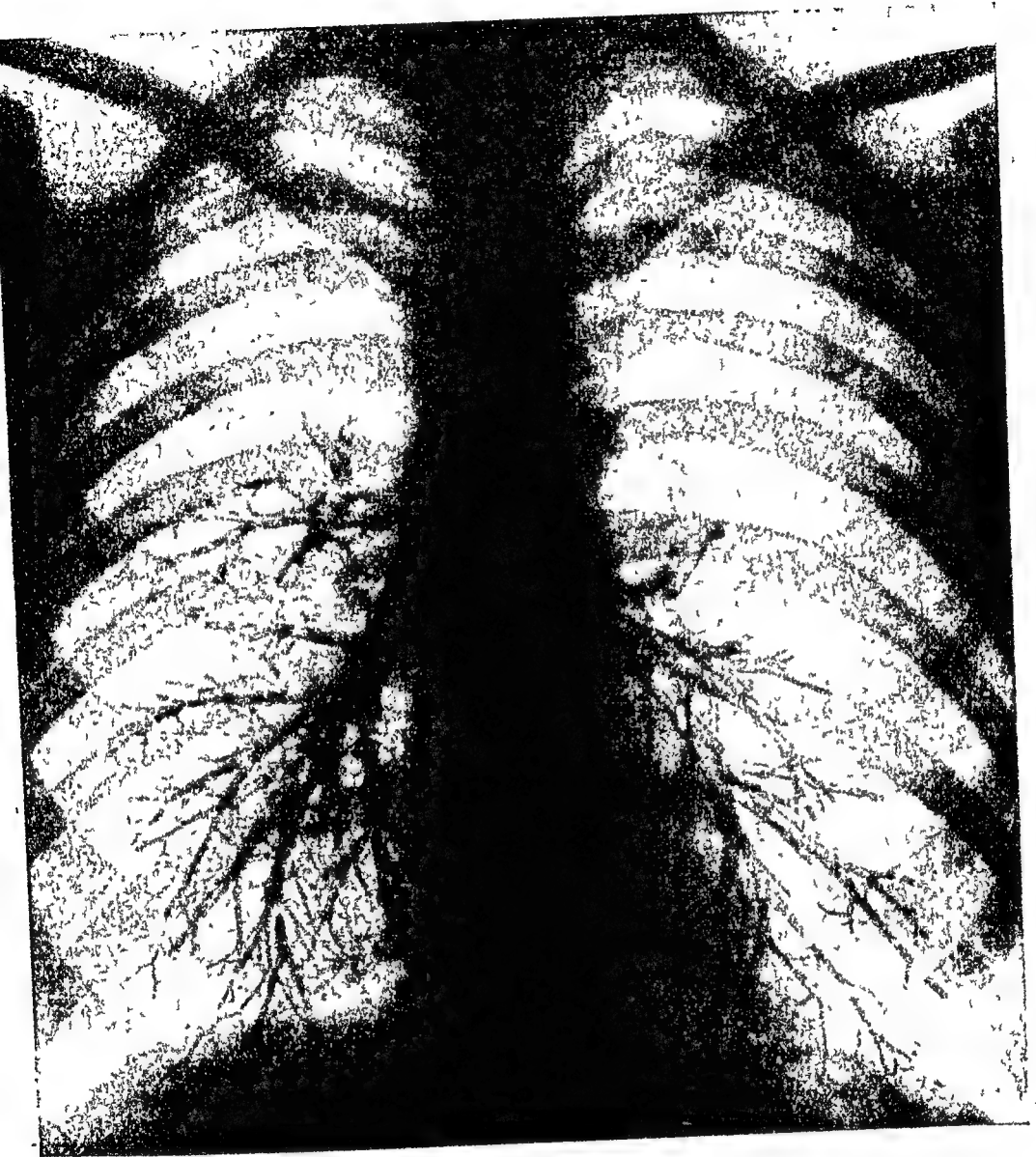


FIG. 100.—Anatomy of bronchi. On right side note oparterial bronchus, apical bronchus to lower lobe, middle lobe bronchus and basal bronchi of lower lobe and retrocardiac bronchus. Left side, upper lobe bronchus dividing into axillary-apical and pectoral stems; the latter overlaps the lower lobe bronchi. (*Courtesy of Prof. Murdoch.*)

The distribution of these branches to the middle lobe is as follows :

- (1) To the outer corner, the superficial mammary cardiac.
- (2) To the outer half of the anterior surface, and lower margin. Outer branch of sternocardiac.
- (3) To rest of inner half of anterior surface. Inner branch of sternocardiac.

The middle lobe branches arch first upwards and forwards—as seen in antero-posterior and lateral views. Their terminations arch downwards. In antero-posterior radiograms they are difficult to distinguish from the backwardly directed “dorsal” apical branch of the lower lobe.

Right Lower Lobe Bronchus.—

Divides into the following :

- (1) The posterior horizontal bronchus (first dorsal, *Aeby*).
- (2) The lesser posterior horizontal bronchus (second dorsal, *Aeby*).
- (3) The posterior basal.
- (4) The axillary basal.
- (5) The anterior basal.
- (6) The retrocardiac.

POSTERIOR HORIZONTAL BRONCHUS (*Ewart*) (first dorsal, *Aeby*).—Originating from the back of the main lower lobe stem, nearly opposite the middle lobe bronchus, this branch courses backwards and a little outwards nearly parallel to the upper dorsal interlobar surface. It divides into a posterior and outer (mid-axillary) division. These supply the upper posterior and outer part of the apex of the lower lobe.

Radiologically this branch is important for several reasons :

(1) The portion of lung which it supplies is sometimes separated off as an accessory lobe. (*Pohl's Lobe*, see page 144.)

(2) Further, it is often the seat of a pneumonic consolidation, which in antero-posterior view is projected on to the hilar region, and therefore often called “central” pneumoma.

(3) It is a common site of abscess of the lung.

(4) It is not uncommon for tuberculosis to commence here, and a cavity may be found in it.

Therefore any gross “hilar” shadow should be carefully investigated in lateral projection in view of these possibilities.

LESSER POSTERIOR HORIZONTAL (*Ewart*), (second dorsal, *Aeby*).—This comes off just below the preceding, and is directed downwards and backwards. (Opposite to it the anterior basal comes off, to be described later.) Its branches descend to supply the superficial part of the back of the lower lobe, in its lower half, not reaching the posterior margin of the lung in the posterior cul-de-sac.

POSTERIOR BASAL.—Divides into outer and posterior branches. Their numerous subdivisions supply most of the posterior half of the base (behind the line which represents a continuation of the trachea as seen in the lateral view), including the posterior part (one-third of the diaphragmatic surface and the posterior lung margin). Above and behind, this region is overlapped by that supplied by the preceding (lesser posterior horizontal).

The outer division supplies the axillary and retro-axillary region of the

lower lobe, posteriorly, and the free edge of the lobe in its outer posterior part. The posterior division takes care of the greater part of the posterior base, being assisted on its medial side by the retrocardiac bronchus.

AXILLARY BASAL.—Consists of a single trunk of origin, which bifurcates into a single outer and a single basal division. The former goes to the upper part of the lower third of the lung on the outer side; the latter supplies the free border on its axillary aspect as far forward as the anterior axillary line.

In radiograms these two branches and their subdivisions may often be distinguished—the outer directed outwards towards the fifth or sixth rib in the axillary line, the basal arching downwards and outwards into the lateral costophrenic recess.

ANTERIOR BASAL.—This arises from the front of the lower lobar stem, is directed downwards and forwards and supplies a wedge-shaped territory limited in front by the lower part of the main fissure, bounded on the outer side by the free surface of the lobe in front of the anterior axillary line. On the medial side the region supplied does not reach the mediastinal surface. This region of the lower lobe is supplied by the retrocardiac bronchus.

RETROCARDIAC.—This small branch is of special interest, since the region it supplies is sometimes separated as an accessory lobe (Retrocardiac, Inferior Accessory Lobe). It arises directly from the inner side of the main stem. It descends vertically and divides into anterior and posterior branches, which supply the posterior part of the inner surface of the lower lobe. Seen from the front, this territory is wedge-shaped, its outer surface sloping downwards and outwards from the hilum. In the radiogram it includes the region between the outwardly sloping posterior basal branches on the outer side and the edge of the spine on its inner side. It is partly overlapped by the right edge of the heart.

BRONCHI OF THE LEFT LUNG

The absence of the middle lobe modifies the bronchial pattern in the left side. The supply to this region must therefore be described.

The Left Main Bronchus.—The left bronchus, larger than the right, is directed obliquely downwards and outwards, making a sweeping curve with convexity downwards. Opposite the inner end of the third anterior interspace it divides into an upper lobe branch, directed upwards, and a lower lobe branch, which continues the line of the main stem, usually overlapped by the left border of the heart. Above the curve of the main bronchus, a shadow is seen with a free crescentic upper margin; this is the left pulmonary artery.

Left Upper Lobe Bronchus

The upper lobe bronchus divides into an upper (axillary-apical) and a lower (pectoral), which almost immediately subdivide.

AXILLARY-APICAL STEEL—This stem divides into *apical* and *axillary* branches. The *apical* branch, at first nearly vertical, runs upwards to supply a territory comprising the anterior and inner third of the lung apex. It is seen

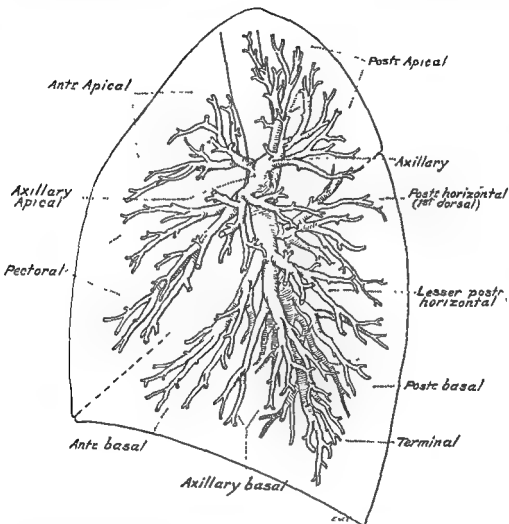


FIG. 101.—Diagram: Bronchi of the left lung, lateral view.

in radiograms to the outer side of the aortic knob. Its branches are found in the first interspace and above the clavicle.

The *axillary* branch divides like that on the right side, into anterior and posterior branches. They are directed upwards and supply respectively (a) the lower or basal zone of the apex in the outer anterior and upper axillary sur-

face, and the posterior and outer part of the anterior surface—with the lung included between them. Its branches are found in the second interspace.

PECTORAL STEM.—This divides into inner and outer branches, both directed forwards. The inner turns inwards towards the heart, and gives branches running downwards and inwards. The outer breaks up into branches which run outwards.

The area supplied by the branches of the pectoral division is roughly equivalent to that of the middle lobe on the right side, but extends a little higher. Thus the outer pectoral sends branches upwards and outwards to the region of the upper lobe, seen in the third interspace, and behind the second rib, and branches running forwards to the supramammary region and fourth interspace in the axillary line. It also supplies the outer anterior part of the lobe down to the main fissure.

The inner (sternopectoral) (fourth and fifth interspaces) supplies the inner half of the lower part of the upper lobe, its pericardial surface, and the lingual tip.

The vessels accompanying the sternopectoral are sometimes seen curving strongly inwards and downwards in the lower left hilar region.

Left Lower Lobe Bronchi.—The bronchi of the lower lobe of the left lung do not need to be described in full detail, since most of the description of the lower lobe bronchi of the right lung applies also to those of the left lower lobe. The main difference here is in the point of origin of the left retrocardiac bronchus, which is a tertiary branch arising from the left anterior basal. Its territory is occasionally divided off as an accessory lobe.

The final division into basal trunks occurs a little higher on the left side than on the right.

The left basal branches are directed a little more outwards than those on the right side. "They form a long flexible spray, drooping with unequal curves."

The apex of the lower left lobe reaches a little higher than the right. Modifications in the direction of the left posterior horizontal (first dorsal) and the lesser posterior horizontal therefore occur.

BRONCHIAL SEGMENTS

The regions supplied by individual bronchi represent definite entities, or segments of the lung. When such a region is consolidated by inflammatory disease (abscess, tuberculosis, localised pneumonia, or pneumonitis) or when the bronchus to this segment is blocked, an area of shadow of pyramidal shape is produced. It is often possible to make an accurate guess as to the segment involved from the plain postero-anterior film, but without the lateral view this guess will be liable to go astray, because it is not always possible to tell whether the shadow is anterior, posterior, or at mid-depth of the chest. The localisation of consolidated areas is of particular importance in abscess of the

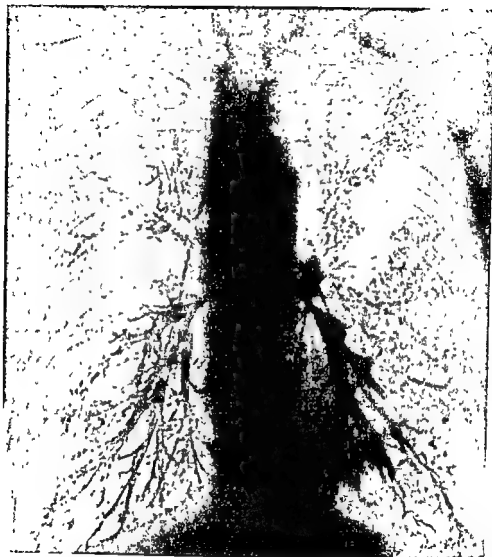


FIG 102 -Lapidol injection to show anatomy of bronchi. On the right side note the eparterial bronchus, middle lobe bronchus, and lower lobe bronchus, with their principal branches. On the left side, the upper lobe bronchi are incompletely filled, the lower lobe bronchi well filled

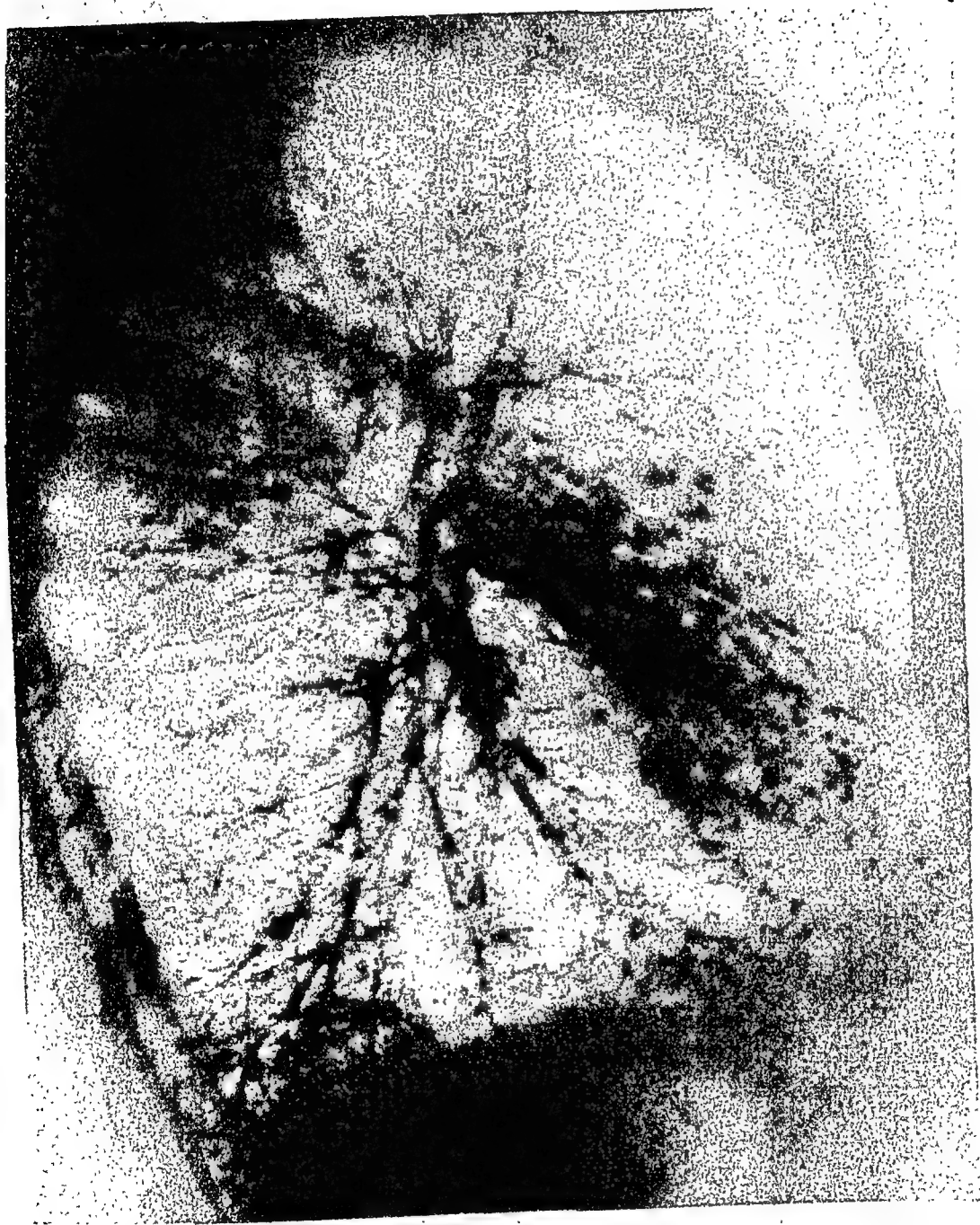


FIG. 103.—Anatomy of bronchi: lipiodol injection. Bronchi of right upper, right and left lower, and right middle lobes in lateral view.

lung, since treatment by postural drainage, by bronchoscopic drainage, or by surgery depends upon such localisation. The area involved points to implication of particular bronchus. The actual shape and position of the consolidated

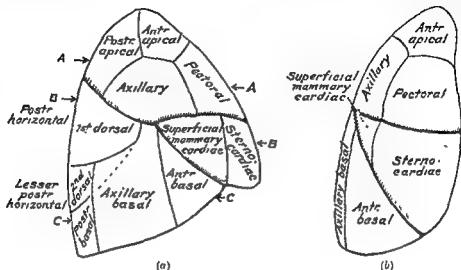


FIG. 104.—Diagram: Bronchopulmonary segments of right lung. The areas mapped out on the surface are the bases of roughly pyramidal segments, having their apices towards the hilum: each is supplied by a single large bronchus. The interlobar fissures are shown by heavier lines. For sections through A-A, B-B, and C-C, see Fig. 103.

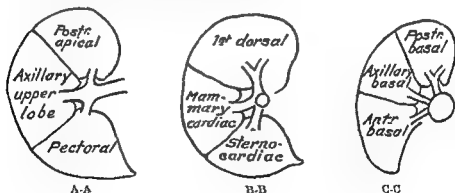


FIG. 103.—Diagram. Sections through the lung at the levels indicated in Fig. 104 (After Glass, modified)

areas may be much influenced by the factors of atelectasis, fibrosis, and compensatory emphysema of neighbouring portions of the lung. When atelectasis is present, the areas of density, especially if near an interlobar fissure, have concave edges, due to the bulging of the unaffected lobes towards the atelectatic

area. It will often be difficult to decide between a localised consolidation and collapse, and a collapse or fibrosis of an entire lobe. For example, a fibrosed right upper lobe will show a triangular apical shadow of the same shape and size

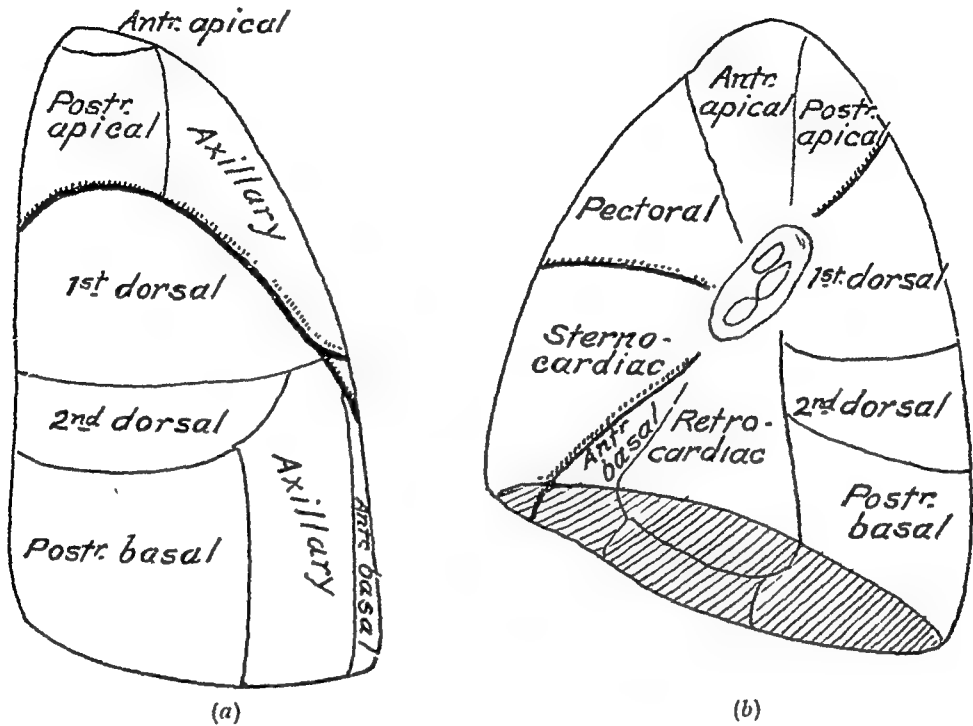


FIG. 106.—Diagram: Bronchopulmonary segments of right lung, (1) seen from behind, (2) seen from mediastinal surface. For explanation see legend to Fig. 104.

as an infiltration in the region of the apical branches of the right upper lobe bronchus: the distinction would depend on the position of the interlobe, which in the former case would form a limiting edge to the consolidated area, and upon the position of the hilum and of the trunk shadows of the middle and lower lobes.

CHAPTER XVII

THE LYMPHATIC SYSTEM OF THE THORAX

LYMPHATIC GLANDS OF THE THORAX

THESE ARE subdivided into parietal and visceral lymphatic glands.

The Parietal Lymphatics are :

(1) **THE STERNAL LYMPH GLANDS.**—These form two groups, each of which lies at the corresponding margin of the sternum along the line of the internal mammary artery. The glands are variable in number (four to eighteen) and in size. They receive afferents from the upper part of the muscles of the abdominal wall, from the diaphragm, from the anterior part of the wall of the thorax, and from the medial portions of the mammaræ. Their efferents communicate with the upper anterior mediastinal glands and with the inferior deep cervical glands, and they terminate on the right side in the right lymphatic or the right broncho-mediastinal duct, on the left side in the thoracic duct. Occasionally, also, they end directly in the internal jugular or the subclavian vein.

(2) **THE INTERCOSTAL LYMPH GLANDS** are lateral and medial. The *lateral* glands lie in the posterior parts of the intercostal spaces, the *medial* are placed in front of the heads of the ribs. Their afferents are derived from the boundaries and contents of the spaces. The efferents of the glands of the upper spaces pass either to the posterior mediastinal glands or to the thoracic duct. Those of the lower spaces on each side form a descending trunk which passes through the aortic opening of the diaphragm and ends in the cisterna chyli.

(3) **THE DIAPHRAGMATIC GLANDS** consist of three sets :

(a) *Anterior* ; (i) behind the xiphisternum, receiving afferents from the liver ; and (ii) lateral, near the seventh costochondral junction, receiving afferents from the anterior part of the diaphragm. The efferents pass upwards to the sternal glands.

(b) *Middle*, on each side, close to the phrenic nerves. On the right side some of these glands are in the pericardial wall in front of the vena cava. They drain the diaphragm, and on the right side the liver. Their efferents pass to the posterior mediastinal glands.

(c) *Posterior* . A few glands at the crura of the diaphragm. They are connected with the lumbar and posterior mediastinal (visceral) glands.

The Visceral Lymphatics are :

(1) **THE ANTERIOR MEDIASTINAL GLANDS**, forming two groups, a lower and an upper. *The lower group* consists of three to four glands, and is situated posterior to the sternum, in the lower part of the anterior mediastinum. It

receives afferents from immediately adjacent parts and from the liver and diaphragm. Its efferents communicate with the upper anterior mediastinal glands, and they end, for the main part, in the broncho-mediastinal trunk. *The upper group* consists of from eight to nineteen glands which lie posterior to the manubrium sterni and anterior to the thymus and the great vessels of the superior mediastinum. Their afferents are derived from the lower group of anterior mediastinal glands, from the pericardium, the heart, the thymus, the thyroid gland, and from the sternal glands. Their efferents pass mainly to the broncho-mediastinal trunk, but they communicate with the medial inferior deep cervical glands, and possibly also with the thoracic duct.

(2) **THE POSTERIOR MEDIASTINAL GLANDS**, eight to twelve in number, lie along the descending part of the thoracic aorta and the thoracic part of the œsophagus. They receive afferents from the diaphragm, the pericardium, the œsophagus, and other immediately adjacent tissues. Some of their efferents join the thoracic duct, others the broncho-mediastinal trunk, and some pass to the bronchial glands.

(3) **THE BRONCHIAL LYMPH GLANDS**.—Under this term are included all the lymph glands which are closely associated with the walls of the intrathoracic part of the trachea and with the main bronchi and their intrapulmonary branches, namely, the

VISCERAL LYMPHATIC GLANDS OF THE TRACHEOBRONCHIAL TREE

The general arrangement of the lymphatic glands in relation to the tracheo-bronchial tree may be studied from the diagrams after *Sukiennikow* (Fig. 107), *Assmann* (Fig. 111), and *St. Engel* (Figs. 108, 110). The exact relationship to the neighbouring vessels has also been described by *Nelson*. They are found massed at the bifurcation of the bronchi, and this arrangement continues throughout the lung up to the third bifurcation. *St. Engel* believes that the glands are more intimately related to the pulmonary vessels than to the bronchi, but agrees that for purposes of description, at all events outside the hilum, the bronchial bifurcation may be used (see Fig. 108).

The following groups occur :

(1) **The Tracheal Group** ("paratracheal") along the trachea.

(2) **The Tracheobronchial Group**, extending downwards into the upper surfaces of the main bronchi. The *right* tracheobronchial group is a large one and lies on the antero-lateral aspect of the trachea, and extends down on to the upper lobe bronchus. The *left* tracheobronchial glands are less constant (*Nelson*). The main group lies on the lateral aspect of the trachea under cover of the aortic arch, but may extend downwards above and also to the outer side of the left main bronchus behind the pulmonary artery. On the right-side these glands vary in number from five to nine, on the left from three to six. Those on the left are in close relation with the left recurrent nerve. Their afferents are derived from other groups of bronchial glands and from the adja-

cent parts of the trachea and bronchi. They are connected with the anterior and posterior mediastinal glands. Their efferents pass to the broncho-mediastinal trunk and also to the inferior deep cervical glands. They are associated, also, by interglandular vessels, with the paratracheal glands.

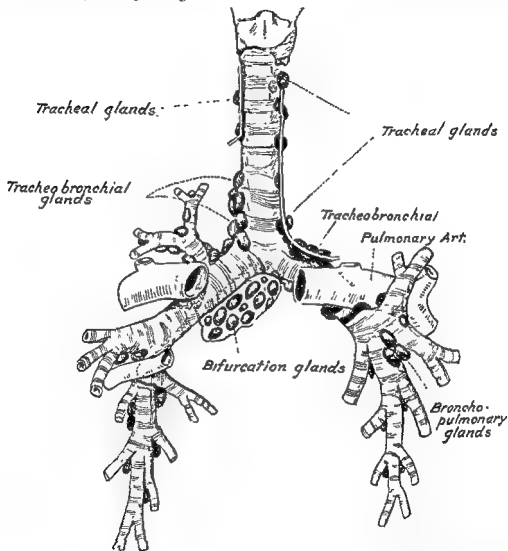


FIG. 107.—Diagram. Lymph nodes of the tracheobronchial tree. (Sulimnikou)

(3) The Bifurcation Group is wedged in the tracheal bifurcation and extends downwards on either side to the commencement of the lower lobe bronchus. The glands are situated between the roots of the great vessels anteriorly and the cesophagus and the aorta posteriorly. Their afferents are derived from the

bronchopulmonary glands and from adjacent parts; their efferents terminate in the tracheobronchial glands. They are connected with the posterior mediastinal glands.

(4) Each group of bronchopulmonary glands, right and left, lies in the hilum of the corresponding lung, in the angles between the branches of the bronchial tube. The glands vary considerably in number, and receive afferents, either directly or through the pulmonary glands, from the lung substance. They also

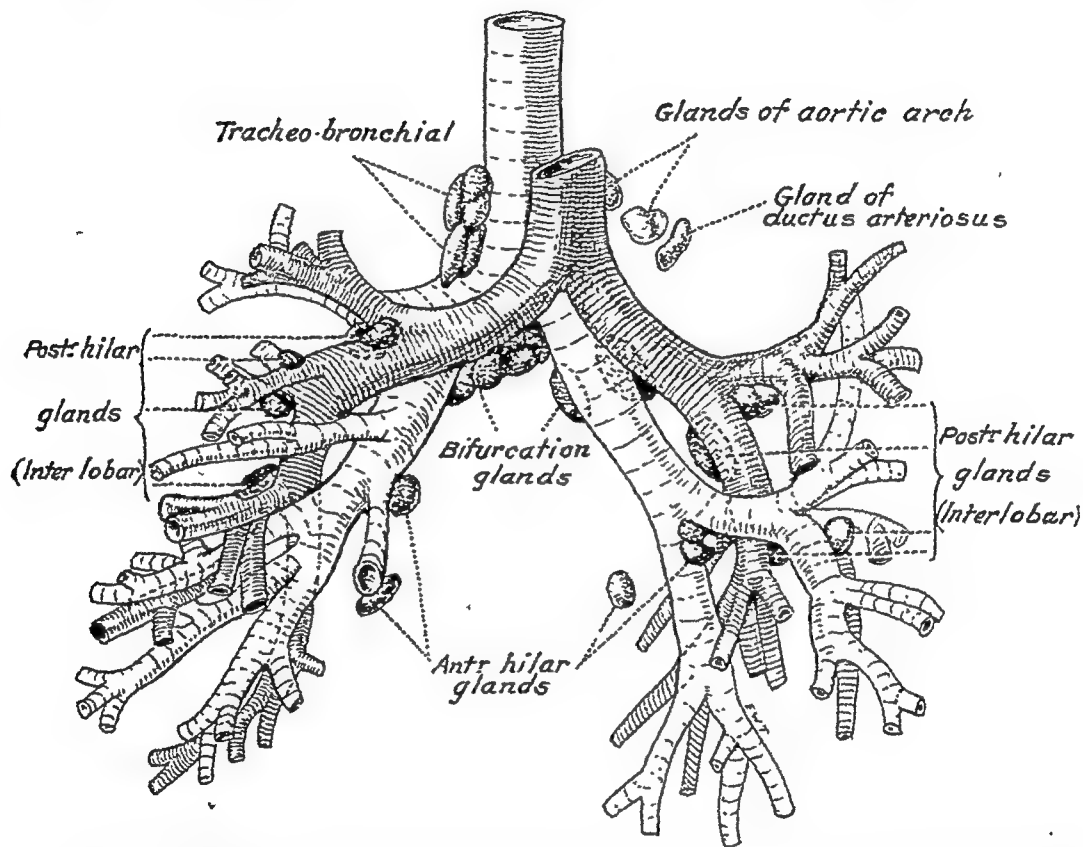


FIG. 108.—Diagram: Tracheobronchial tree, and pulmonary artery, showing relations of the lymph nodes to them according to St. Engel. (*Handbuch d. Roentgendiagnostik im Kindesalter.*)

receive afferents from the pleura; their efferents pass to the tracheobronchial glands and to the glands of the bifurcation.

(5) The pulmonary lymph glands lie in the lung substance, usually in the angles between two bronchial tubes, up to the third branching. Beyond this point the existence of true lymph glands is very doubtful, although it is stated by some anatomists that these are to be found as far out as the fourth or fifth branching. Probably there are, at most, lymph nodes consisting of aggregations of lymphoid cells, and it may be that even these are not normal

structures, but a response to infection or other stimulus, and therefore to be regarded as pathological formations.

The afferents of the pulmonary glands are derived from the lung substance, and their efferents pass to the bronchopulmonary glands.

St. Engel divides the hilar glands into two groups, an anterior (bronchopulmonary) and a posterior, in close conjunction with the interlobar spaces, and separated from them only by a thin layer of pleura. These, as *Fleischner* has observed, are liable to infect the interlobar pleura and give rise to interlobar pleurisy. The lymphatic glands of the left upper lobe also show a peculiarity,

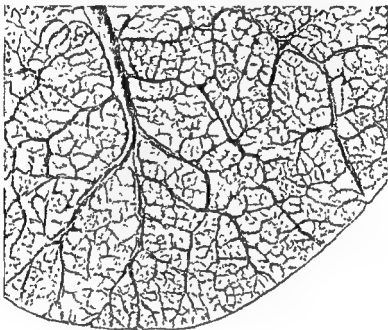


FIG. 103.—Drawing of lower surface of middle lobe, showing lymphatic vessels outlining the lobules, and acini of the lung parenchyma. About twice natural size.

according to *Engel*, in that some of them lie not in the lung root, but in the anterior mediastinum in close relationship to the aorta and ductus arteriosus (see Fig. 108).

The glands associated with a given lobar bronchus receive the deep lymphatic vessels from the corresponding lobes, running with the bronchial and pulmonary vessels. A lesion in a given part of the lung, therefore, drains into a specific group of bronchial and hilar glands. The middle lobe drains into both the upper and the lower lobe groups. The hilar glands also receive lymphatics from the visceral pleura by lymph vessels which drain along the interlobar fissures and over the anterior and posterior lung surfaces, towards the hilum.

The superficial and the deep lymphatic system intercommunicate on the surface of the lung. The important lymphatic pathways along the interlobar fissures account for the frequency with which these become visible in the radiogram in pulmonary disease, inflammatory or neoplastic, and in some pleural infections.

ENLARGED LYMPHATIC GLANDS

In searching for the glands in a radiogram, the bronchi must be used as a guide, and these may be picked up, especially in children, as translucent streaks. The tracheal and tracheobronchial glands may be readily recognised, since they tend to project above the hilar shadow. Those at the bifurcation are rarely seen

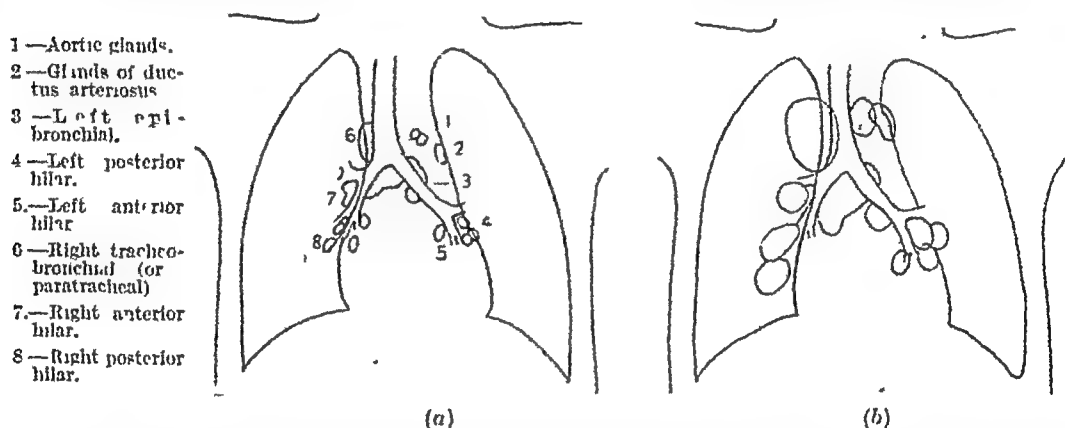


FIG. 110.—(a) Lymph nodes in normal state. (b) Same lymph glands enlarged. (After St. Engel.)

unless calcified, when they may occasionally be found in oblique or lateral views. The hilar glands are usually difficult to pick up, firstly because they are overlapped by the heart shadow, and secondly because they are covered by the pulmonary artery and its branches and other components of the hilar shadow, so that rounded nodes in the actual hilum are not always seen; the picture during the stage of acute inflammatory change is rather that of a diffuse thickening of the lung root ("massive hilar shadow"). The difficulty is to decide between the various processes which may give rise to this diffuse shadow, namely (a) stasis in the vessels; (b) bronchitic and peribronchitic inflammation; (c) lymphadenitis and perilymphadenitis; and (d) pulmonary inflammations situated in front of or behind the hilum, the shadows of which fuse with the hilum in a postero-anterior view.

Certain groups of glands are so placed as to be easily seen when enlarged, even though uncalcified (Figs. 110, 111). Such enlargements can be due to tuberculosis, neoplasm, influenza, pneumonia, other acute respiratory diseases, especially in children, to anthracosis or pneumoconiosis. These are:

(1) The right paratracheal glands.

(2) The left tracheobronchial glands between the aortic knob and the left pulmonary artery.

(3) The right tracheobronchial glands lying above the right pulmonary artery in close relationship to the eparterial bronchus.

(4) The right bronchopulmonary (hilar) glands, when these lie to the outer side of the pulmonary artery or are sufficiently enlarged to project beyond it. The left bronchopulmonary (hilar) glands are less well placed for observation, being often hidden by the left border of the heart and conus of the pulmonary artery.

(5) The retrosternal glands.

The bifurcation glands at the tracheal bifurcation are rarely seen, being overlapped by the heart, mediastinum, and spine. Occasionally, when calcified, they are visible in postero-anterior films taken with sufficient penetration, and may sometimes be seen in oblique or lateral views. *Assmann* has recognised rounded shadows in this situation due to glandular metastases from carcinoma of the oesophagus. The observation was verified at autopsy.

Appearance of Enlarged Mediastinal and Hilar Glands

These project into the lung field as rounded shadows, and the separate masses of glands show as individual projections, giving a scalloped contour to the whole shadow. This appearance is typically seen in children with tuberculosis. It is also sometimes seen in lymphadenoma or new growth.

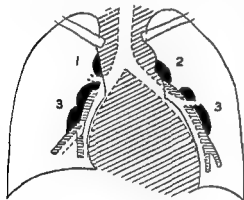


FIG 111 —Diagram of the lymph nodes most frequently seen in radiograms when large enough to project from the mediastinal shadow. 1 Right paratracheal 2 Glands between aortic knob and pulmonary artery 3, 3 Bronchopulmonary glands in the hila (After *Assmann*)

Coarse strands sometimes radiate from them into the lung field, due, probably, to stasis in the lung vessels. In less acute cases these strands are denser, and in chronic cases have been shown to be due to peribronchial and perivascular fibrous tissue formation. The well-defined contour of enlarged inflammatory glands is often obscured in children by peradenitis, or localised pneumonitis of the surrounding lung. Occasionally, the writer has recognised in the hollow-back view evidence of concomitant interlobar

pleural inflammation, due to the close relationship of the posterior hilar glands to the interlobar pleura, described by *St. Engel*.

Small glands lying in the angles of the bronchi at the edges of the hilar shadow, especially those related to the eparterial bronchus and left upper lobe bronchus, may be simulated by branches of the pulmonary artery seen end-on. They may be distinguished by the following points :

(1) The end-on arterial shadows occur along the course of the trunk shadow, and each has the same diameter as the trunk on which it occurs. The end-on vessels diminish regularly in diameter towards the lung.

(2) By turning the patient slightly to one side or the other, the end-on vessels again become linear.

It is often advantageous to examine the chest in children with the patient slightly rotated in this manner, in order to project enlarged glands clear of the heart shadow.

Calcified Glands

When calcified, glands show a typical granular density on the surface and in their substance. Old glands may form dense homogeneous patches. Careful inspection of a group of hilar glands will often localise them to a definite bronchus, and leading outwards along that bronchus smaller glands may be seen, directing the eye towards a definite region of the lung in which lies a calcified primary parenchymatous lesion, which may be minute (Ghon's focus). The focus and the glands in its drainage area are evidence of an old puerile tuberculous lesion (Ranke's complex) which has healed.

ENLARGEMENT OF GLANDS IN LYMPHADENOMA AND MALIGNANT DISEASE

The tracheal and tracheobronchial glands are most evident in these conditions, and in lymphadenoma it is the upper mediastinal glands which are mostly affected and most massive; but a hilar form may occur, usually bilateral. The contour may be smooth and regular or may have a lobulated appearance due to enlargement of individual glands. Enlarged hilar glands often fit neatly into the concavity of a main bronchus; this is particularly well shown in involvement of the left tracheobronchial group, in which the left upper lobe bronchus is seen curving downwards beneath the gland. In this case curved strands of density directed upwards to the apex sometimes give the shadow a fuzzy border. This appearance, which the writer has noted on many occasions, may possibly be due to pressure on the left superior pulmonary vein and a consequent stasis. This vein, draining the left upper lobe, passes downwards in front of the left upper lobe bronchus and left main bronchus near its termination; its right border is separated from the right pulmonary artery by an interval of 1 cm., and it terminates at the level of the bifurcation of the common pulmonary artery (*Ewart*). The vein is therefore in such a position that it may be obstructed by any considerable glandular enlargement of the left tracheobronchial or upper bronchopulmonary lymphatic vessels. According to *Assmann* it is also obstructed by the enlarged pulmonary artery in mitral stenosis, producing a similar but less-marked appearance of increased striation in the left upper lobe. It is possible for an aortic aneurysm to produce a similar

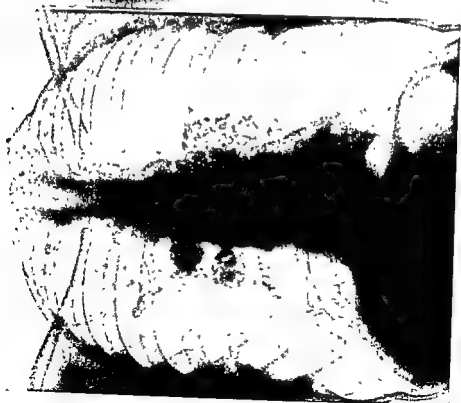
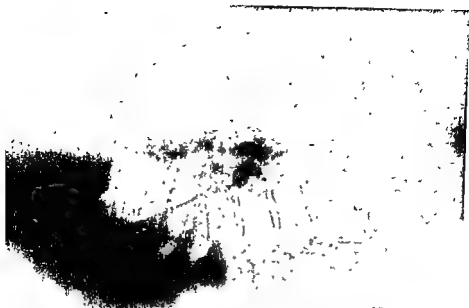


FIG. 112.—Calcified lular and cervical glands



Lateral view.

appearance, and this the writer has observed. In lymphadenoma the retro-sternal glands are often enlarged, and show clearly in the lateral view as a shadow sometimes as large as an orange in the interval between the sternum and the great vessels.

LYMPHATICS OF THE LUNG

Lymphatic Vessels.—These, according to *Miller*, are found in the lung parenchyma in two main groups: (1) perilobular lymphatics around the veins in the interlobular septa; and (2) intralobular lymphatics around the terminal bronchioles and bronchial arteries. These two groups of lymphatic vessels communicate within the lobules. The perilobular lymphatics also communicate with the subpleural lymphatics (see Fig. 103). The figure shows in addition the sites at which *deposits of lymphoid cells* are found: (1) at the junction of the perilobular and pleural lymphatics; (2) in relation to the intralobular veins at their junction with perilobular veins; (3) at the root of the lobule in the bifurcation of the pulmonary arterial or alveolar duct. These lymphoid deposits are believed to be the site of origin of the characteristic nodules of pneumoconiosis.

As regards the further course of the lymphatics in the lung, between the periphery and the hilum, little is known. They certainly run in the lung stroma in association with the bronchi and vessels. *Sinason* and *Strathan* state that they are closely related to the blood-vessels and not interrupted by lymph nodes between periphery and hilum. They think they are functionally related to the air tubes only. The distribution of lymphoid tissue is strictly related to the respiratory tract, and not to the vascular system, except where air passages and blood-vessels come into juxtaposition, and even here the association is anatomical and not physiological.

LYMPHATIC PATHWAYS OF THE MEDIASTINUM

The thoracic duct receives at the cisterna chyli afferents from the right and left upper lumbar glands and from the mesenteric glands. In the thorax it receives intercostal and mediastinal tributaries and communicates with the right costo-mediastinal trunk. The latter receives the lymph from the right lung and empties via the right lymphatic duct into the commencement of the innominate vein. Near their terminations, both thoracic ducts lie close to the supraclavicular (lower deep cervical) glands. Variations are not uncommon. The thoracic duct may be right-sided or duplicated, or broken up into a plexus of vessels in front of its course. It has many large collaterals. Disease of the thoracic duct, reaching it from neoplasm of the gastro-intestinal tract, or genital system (via lumbar glands), frequently extends to its tributaries. *Winkler* (cit. *Willis*) noted enlarged mediastinal glands and cancerous lymphatics due to direct spread, but in nearly all cases cancer emboli from the thoracic duct reach

the lung by a different route, namely via the innominate vein, right heart, and pulmonary artery, and are therefore blood-borne emboli. Another occasional route by which abdominal carcinoma may spread to the lung is from direct invasion of the hepatic veins by metastatic deposits in the liver, a final dissemination occurring through the pulmonary arteries. In either condition the lung metastases may take the form of a few large nodules or, rarely, of miliary carcinomatosis.

The lymphatics of the breast are connected with the retrosternal and anterior mediastinal glands through the anterior intercostal spaces, and may be directly infected from carcinoma of the breast. In this condition evidence of enlargement of these glands is rarely seen in radiograms, which show enlargement of the hilar and tracheobronchial glands, with little evidence as to the route by which the disease has reached these glands. From the glands in the hilum the disease may extend outwards into the lung by permeation along the pulmonary lymphatics (lymphangitis carcinomatosa).

Bronchial or œsophageal carcinoma may invade the posterior mediastinal glands by direct extension in the lymphatics, and the writer has seen several instances in which the spine and ribs have been locally invaded by this route. the bone lesions were limited to the vertebræ and ribs at the level of the primary lesion

If the leaves of the pleura are adherent, direct continuity between the visceral and parietal layers of pleura may allow the spread of infection or neoplasm from the lung to the intercostal spaces and ribs.

CHAPTER XVIII

UPPER AIR PASSAGES

PHARYNX AND EPIPHARYNX

Foreign Bodies occasionally lodge in the pharynx or epipharynx. The writer has seen a safety-pin impacted in the epipharynx in an infant. It is therefore necessary to include this region in fluoroscopic examination.

Adenoid Vegetations narrow the epipharynx, and their position and extent may be well shown by X-ray examination. In the case of adenoids *Groth* (1934) advises this method in preference to palpation, as being less disturbing to the child and as giving a better idea of the degree of obstruction of the air passages. *Grandy* was the first to use this method, which he described in a short communication in 1925.

Malignant Tumours of the Nasopharynx also encroach upon the air space, and may be detected by X-ray. They may also invade the base of the skull; the writer has seen several instances of such invasion with cerebral symptoms or exophthalmos. Basal radiograms of the skull in the vertical projection showed definite erosion in the middle fossa.

Tumours in the Epipharynx may be outlined by instilling lipiodol into the nose and making lateral radiograms with the head in the inverted position. The patient lies supine with the head hanging backwards (*Zupping*).

LARYNX

The larynx does not offer great scope for X-ray examination, though lateral views with low penetration are useful in malignant diseases and ulcerations of the larynx.

Normal Larynx

IN THE THYROID CARTILAGE calcification is normal, and increases with age. In the female calcification is usually confined to the cricoid and posterior border of the thyroid cartilage. In the male a second centre is formed which spreads along the lower and anterior margins of the thyroid cartilage. From this an oblique bar may extend upwards and forwards in the ala towards the upper border. Oval clear areas ("fenestræ") are enclosed between this bar and the other borders (*Bjorn Warnung*). These clear areas are normal, and must not be mistaken for "erosion" by laryngeal tumour. Finally the alæ become completely calcified. The calcification is eventually converted into true bone. The writer has occasionally observed calcification in the cartilagine triticeæ

above the superior thyroid cornua in the thyrohyoid ligaments. They may be mistaken for a "foreign body."

In antero-posterior radiograms the alæ of the thyroid cartilage are often visible, directed obliquely downwards and inwards, projected upon the cervical spine.

THE CRICOID CARTILAGE ossifies early in the female. Often the posterior edge alone is visible, and the vertical line or patch of calcification in it has often



FIG 113 — Calcified laryngeal cartilages (thyroid and cricoid)

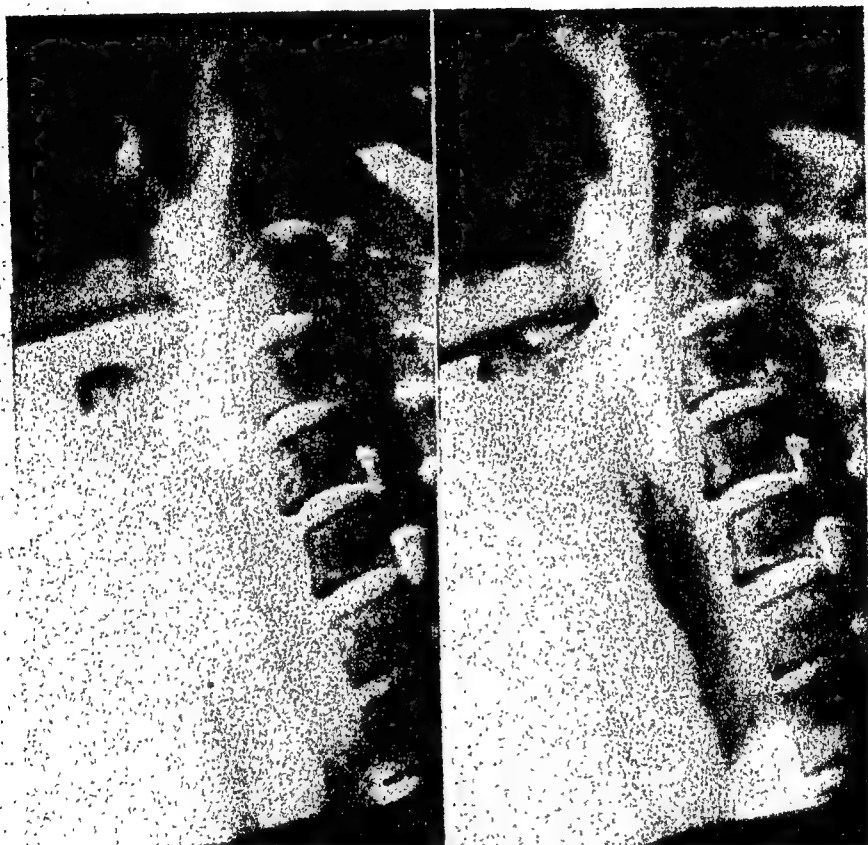


FIG 114 — Normal larynx F. 21. Lateral view. Small calcification in cricoid cartilage. Note epiglottis, vallecula, arytenoid epiglottic folds, and sinus of Morgagni

been mistaken for a swallowed foreign body. The distinction is easy if barium is given and carefully watched. It passes *behind* the calcification.

THE CAVITY OF THE LARYNX.—In lateral "soft tissue" radiograms the cavity of the larynx is seen as a clear space bounded above and in front by the curved shadows of the epiglottis and the anterior wall of the vestibule, below by the horizontal translucent streak of air in the sinuses of Morgagni. Above the sinus of Morgagni a faint horizontal band of shadow indicates the position

of the false cords. If the posterior surface of the epiglottis is traced downwards, the aryteno-epiglottic folds are seen as a shadow running obliquely downwards and backwards towards the shadow of the cricoid. Visualisation of the larynx is improved if the hypopharynx is distended with air by an expiratory effort with closed mouth and nostrils (*Jönsson*). This manœuvre may succeed in separating the posterior border of a hypopharyngeal tumour of



(a)

(b)

FIG. 115.—Normal larynx and trachea, (a) at rest, (b) distended by expiratory effort with closed mouth. Note slight bulging of posterior wall of trachea, and opening up of the hypopharynx and laryngeal vestibule in (b).

the anterior wall from the retropharyngeal tissue, so defining its margin (*Zuppinger*). The chin should be slightly elevated. The writer finds that the valleculæ and epiglottis are more clearly shown if the tongue is protruded between the teeth.

Malignant disease or extensive non-malignant ulcerations may cause (1) traction of the epiglottis, pulling it downwards and backwards, opening up the valleculæ; (2) visible thickening of the epiglottis or aryteno-epiglottic folds;

(3) partial obliteration of the clear space of the laryngeal cavity due to tumour; (4) obliteration of the sinus of Morgagni; (5) increased shadowing of the false cords, (6) destruction of the laryngeal cartilages. Such studies may assist in determining the downward extent of the growth behind the larynx

but require very careful correlation with laryngoscopic findings.

Polypoid Tumours of the Larynx.—Four cases of polypoid tumour of the larynx, outlined in a lateral radiogram, have been demonstrated by *McGehee*.

Laryngocele.—A round air-containing cavity in the neck, lateral to the larynx, is due to laryngocele (laryngeal cyst). This rare condition has been described by *Bland Sutton*. In the chimpanzee the deep cervical fascia is undermined by diverticula of the laryngeal mucous membrane. This communicates with the larynx through the thyrohyoid membrane. This air cell is especially large in the howling monkey. *Bland Sutton* mentions no case in man, but the writer has seen two



FIG. 110.—Laryngocele. Air-containing cavity communicating with the larynx. F. 40. Autopsy

radiograms showing a round air cyst in the neck, apparently originating in this way. One of these was observed by Prof. Woodburn Morrison, who first drew the writer's attention to the condition. The second was a personally observed case.

TRACHEA

Anatomy.—In the postero-anterior radiogram the commencement of the trachea is seen at the level of the cricoid (sixth or seventh cervical vertebra).

Traced upwards, it rapidly tapers to a point at the glottis. Traced downwards, it is seen as a clear stripe which ends in the bifurcation at the level of the sixth to eighth dorsal vertebra. Its length is 12 cm. in the adult male; 7 cm. in a child of 10 years; 4 cm. in an infant. Diameter—male 15–22 mm.; child 8–11 mm.; infant 6–7 mm. It moves slightly upwards on swallowing and downwards with inspiration as much as 1 cm.

Calcification of Trachea.—In lateral views calcification in the tracheal rings is evidenced by nodular shadows along the anterior margin. Occasionally the whole ring is calcified, showing transverse stripes across the trachea in the lateral view. A chondroma may originate in a tracheal ring and has been recognised radiologically.

Deformities of the Trachea.—With the rare exception of syphilitic stenosis, all tracheal narrowings are due to extrinsic compression. Retropharyngeal goitre, a rare condition, compresses the trachea from behind; retrosternal goitre compresses it laterally and anteriorly. An indentation of the posterior wall may be due to tumour in the œsophageal wall. Anterior displacement may also result from retropharyngeal abscess. Caries of the spine or foreign body should be looked for. Pharyngeal diverticulum may displace the trachea forwards; in cases of right-sided aorta both œsophagus and trachea are displaced forward and to the left.

A characteristic deformity of the trachea has been described by *Pancoast* and *Pendergrass*, due to an enlarged thymus in infants.

Above the manubrium, bilateral compression of the trachea by thyroid enlargement (simple or malignant) is a common finding (scabbard trachea). If the goitre is unilateral, the trachea is more deformed on that side, and often displaced and strongly curved towards the opposite side. The lower end of the trachea is normally displaced slightly to the right by the aorta. Lateral displacement of the trachea is an important physical sign. It is seen commonly in tumours of the thyroid gland, cervical glands, malignant glands in the mediastinum, aneurysm (aorta or innominate), pneumothorax, pleural effusion, fibrosis of the lung, and atelectasis. In severe scoliosis the trachea lies to one side of the midline (on the side of the concavity). Lateral displacement of the trachea from this or any other cause is sometimes sufficient to produce abnormal physical signs on that side and changes in the respiratory sounds which may be misinterpreted.

The most characteristic deformity of the trachea is an angulation, kinking, or curvature towards a contracted apex. This occurs most frequently as a result of fibrosis, usually tuberculous. It has been stated that curvature towards the affected side does not occur as a result of neoplasm with atelectasis of the upper lobe. This is incorrect; it does occur, though less frequently and to a less marked extent than with fibrotic lesions of the apex. Localised dilations of the trachea from scar contracture may occur (*Fleischner*, cit. *Schinz*) and tracheal softening, according to *Sgalitzer* and *Stöhr*, may be demonstrated

by fluoroscopy; if the patient makes a strong expiratory effort with closed glottis, the softened wall bulges outwards; if he makes a strong inspiratory effort with closed glottis, the wall bulges inwards. The condition may follow long-standing tracheal compression.

A splaying out of the tracheal bifurcation by an enlarged left auricle has been observed in mitral disease.

It cannot be sufficiently emphasised that very soft postero-anterior radiograms are not suitable for diagnosis of tracheal conditions. Sufficient penetration should be used, particularly in the case of aneurysms or tumours of the upper mediastinum, to show the trachea and main bronchi clearly. The Potter-Bucky diaphragm or Lysholm Grid should be freely used. Tomography is even more valuable for this purpose.

Intratracheal Tumours are rare, and are nearly all found by endoscopy. The associated dyspnoea brings them directly into the province of the laryngologist. Papillomata of the trachea are described by *Bentel*, who published a case outlined by lipiodol. The appearance is that of rounded projections into the lumen, partially outlined by surface films of lipiodol—not unlike that of oesophageal varices. A case of intratracheal tumour is described by *Weiss* and *Biermann*. A rounded tumour shadow was shown in the trachea just above the bifurcation in an oblique view.

Diverticula of the trachea occur. These are usually multiple, but may be single. *Kahler* (cit. *Schinz*), and *Morlock* and *Pinchin* have described this rare condition, which affects the trachea and bronchi. In the case shown by *Morlock* and *Pinchin*, bronchoscopy showed throughout the trachea and larger bronchi, which were dilated, depressions in the intervals between the cartilaginous rings. These were found after lipiodol injection to lead to diverticula from the trachea and bronchi. The largest was the size of a shilling. Most were considerably smaller. The writer has seen one case of isolated diverticulum outlined by lipiodol. Tracheal diverticula always occur near the junction of the trachealis muscle with the semi-rings of the trachea. *Rokitanski* ascribed them to chronic catarrh, *Gruber* to retention cysts of the tracheal mucous glands (*Stoloff*).

Oesophagotracheal Fistula.—The barium may pass through a fistula into the trachea eroded by growth of the upper oesophagus. A more common mode of entry of bismuth into the larynx and trachea is, however, impairment of the swallowing mechanism as a result of infiltration or nerve implication. It is not unusual to see small drops of bismuth pass through the glottis in cases of laryngeal or post-ericoid tumour. The writer recently carefully observed a case of high oesophageal obstruction due to carcinoma, in which barium entered the trachea. It was observed that this did not occur until the level of the barium in the dilated oesophagus was raised by repeated sips to the level of the posterior aperture of the larynx. It then spilled over the arytenoids into the laryngeal cavity and, passing through the glottis, outlined the whole trachea.

FOREIGN BODIES IN THE AIR PASSAGES

Aspiration of foreign bodies into the trachea occurs most often in children. These are rarely large enough to become impacted in the trachea. If they are so impacted, they tend to lie in an antero-posterior plane in contradistinction to those in the œsophagus, which lie transversely. They usually pass onwards into the bronchi; in four-fifths of the cases, into the right stem bronchus. They are nearly always found in a lower lobe bronchus, almost never in an upper or middle lobe. The objects found are such things as children play with, and are likely to put into the mouth: screws, pins, beads, buttons, paper clips, etc. In a large number of cases they are metallic and are recognised in the plain radiogram.

It is stated by *Schinz* that two-thirds of inhaled foreign bodies are non-opaque. This proportion was observed in a series of forty-two cases examined at his clinic. Even metallic foreign bodies may be hidden by ribs, heart, or spine. Iron foreign bodies may leave a local siderosis after the offending foreign body has been coughed up.

Teeth and fragments of tooth are sometimes inhaled, particularly after dental operation, and the recognition of a tooth and its localisation in a bronchus is by no means as easy as in the case of other foreign bodies. The novice may easily be misled into mistaking any dense shadow in the hilar region—for example, a calcified gland, or even an end-on vessel—for the suspected tooth fragment. It is essential to establish the clear relationship of the suspected foreign body with the bronchus in more than one plane. Lateral and oblique views are, therefore, imperative, with suitable penetration. The Potter-Bucky or Schonander grid must often be used. The anatomy of the bronchi must be visualised and kept in mind. Sometimes a foreign body moves its position after the X-ray has been taken, and may be found in another part of the bronchus, in a different bronchus, or in the other side of the chest.

Complications of inhaled foreign bodies are atelectasis, purulent bronchitis, bronchiectasis, pneumonia, lung abscess, and empyema. In atelectasis, the collapsed area corresponds to the distribution of the bronchus involved. It may, therefore, involve a whole lobe or part of a lobe, according to the situation of the foreign body and the surrounding mucosal swelling which blocks the bronchus, so that in a case in which the actual bronchial wall cannot be seen a knowledge of the bronchial anatomy will enable the foreign body to be localised from the position of the atelectasis.

Non-opaque Foreign Bodies

Manges has described in detail, in several valuable papers, the changes which may be observed after inhalation of non-opaque foreign bodies. Non-opaque foreign bodies are nearly always vegetable, including nut kernels and seeds. They produce more inflammatory reaction than the metallic. This reaction may come on in a few days, or after a long interval after inhalation, and is more marked in children under 2 years. About 2 per cent. are coughed up spontane-

ously. The symptom of onset is, as a rule, a "strangling attack" due to glottic spasm, but when the foreign body settles down into a bronchus there may be a quiet period at first. If there is much obstruction, the patient may show well-marked dyspnoea, particularly if the foreign body is in the trachea.

Four definite types of change take place in the lungs. (1) Obstructive emphysema; (2) atelectasis; (3) drowned lung; and (4) lung abscess.

(1) **OBSTRUCTIVE EMPHYSEMA.**—Valvular obstruction is present which is greater in expiration than in inspiration. Air enters the lung more freely than it can be expelled and the lung is over-distended. If the foreign body is in the trachea, both lungs are over-distended. If in the main bronchus, the signs are: (a) increased transparency of the affected lung; (b) diaphragm depressed and somewhat limited on the affected side; (c) heart and mediastinum displaced to the unaffected side on expiration. If in the trachea, the signs are: (a) increased transparency of both lungs; (b) diaphragm depressed and limited; (c) heart rotated and its transverse diameter less on expiration than on inspiration. The presence of obstructive emphysema is always more striking at the end of expiration than in full inspiration. Films should be taken in both phases.

Regarding the displacement of the heart and mediastinal structures to the unaffected side on expiration, if the obstruction is in the lower half of the chest, the heart seems to swing as if it were a pendulum. If the upper mediastinum is noticeably displaced, it suggests that there is also obstruction of the upper lobe bronchus. Diaphragm movements in tracheal obstruction are sometimes paradoxical, owing to the powerful contraction of the accessory muscles.

Regarding the rotation of the heart, the apex comes towards the anterior chest wall. In the normal condition the transverse diameter of the heart is greater on expiration than inspiration. The reverse of this has been found a valuable sign in obstructive emphysema due to foreign body in the trachea. The signs in any given case may be very variable, owing to the foreign body changing its position.

It must be remembered that obstructive emphysema may, in rare instances in children, be caused by the pressure of enlarged bronchial glands upon a bronchus.

(2) **ATELECTASIS.**—Collapse of the lung distal to the foreign body. This occurs when the obstruction is complete, so that air can neither enter nor leave. In this case the air is rapidly absorbed from the alveoli. The affected side is opaque and the mediastinal structures displaced towards that side. Later the alveoli may fill with pus and exudate, and the diminution in the volume of the lung is then less obvious. Similar atelectasis may occur in bronchial tumours from external pressure on the bronchi by tumour or peribronchial glands. Massive collapse of the lung may be considered one form of foreign body obstruction from which the "foreign body," instead of having been aspirated, is mucus collected in the bronchus at the site of obstruction.

(3) **DROWNED LUNG.**—This is a condition in which exudate arising distal to the foreign body gradually fills the small bronchi and alveoli

by gravity. It rarely occurs alone; it is usually accompanied by some atelectasis. It takes the shape of the portion of the lung supplied by the affected bronchus. Its density is said to be less than that of a lung abscess or

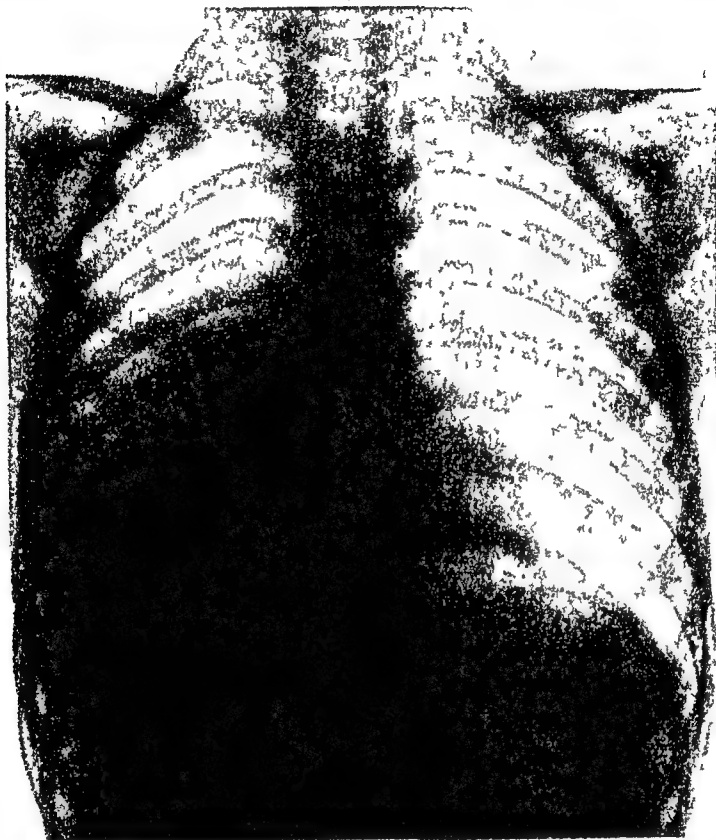


FIG. 117.—M. 7. Atelectasis of right lower lobe due to inhalation of grass seed. This was coughed up and chest rapidly became clear.

consolidation, unless atelectasis has occurred. At first mucous, it later becomes mucopurulent, and finally may become definitely purulent.

(4) LUNG ABSCESS.—Lung abscess may occur early, but is usually a late phenomenon.

In cases showing atelectasis or drowned lung, the location is immediately apparent if the bronchial anatomy is thoroughly understood. This assists the bronchoscopic search. It must be remembered that foreign bodies may change their positions during bronchoscopy.

Radiographic exposures should be made as nearly as possible at full inspiration and at the end of expiration, and it may be necessary to make a child cry in order to get deep breathing. If, however, there is marked dyspnoea, the examination must be carried out as quietly as possible. Struggling has resulted in sudden change in the position of the foreign body and immediate death.

CHAPTER XIX

DISEASES OF THE BRONCHI

THE LARGER bronchi may be seen near the hilum as clear stripes, or, when viewed end-on, as circular rings with a clear centre. The smaller bronchi are not readily visualised in a radiogram, and play but a small part in producing the network of lung shadows. The walls of the bronchi may be visible in the lung parenchyma when calcified in elderly subjects, but normally their ramifications in the lung cannot be traced with certainty, though here and there they may be seen as linear translucent bands cutting across the pattern of the vessel markings. If the walls are thickened, the lumen widened, or the bronchi filled with pus, mucus, blood, or serum, they become in some degree visible; they are often conspicuously visible as translucent bands when surrounded by an area of solid lung. This, as *Fleischner* has pointed out, is a valuable point in the differential diagnosis of pneumonic lung from interlobar effusion. Bronchi seen against a background of effusion are not rendered more visible thereby, but when surrounded by consolidated lung they show up by contrast. Bronchi when dilated become visible in bronchiectasis, or apical bronchiectasis in fibroid tuberculosis.

BRONCHITIS

In bronchitis the bronchi are visible when filled with secretion. This usually occurs in *acute bronchitis*; there may also be some temporary atelectasis of the lung lobules, giving a fine mottled appearance which in an uncomplicated case rapidly clears up. *Saul* illustrates such a case in a young man radiographed during the early stages of an attack of influenza. This appearance is really due to a bronchiolitis.

Bronchitis deformans is due to pressure of a lymphatic gland upon a bronchus. In a case described by *Saul* it was an upper lobe bronchus; the bronchial branches concerned were visible as streaks radiating from the hilum. Secretion within them was the probable cause of the opacity.

Saul believes that the increase in the linear shadows of the lung in bronchitis is due entirely to the secretion in them and not in any degree to "peribronchial" thickening, mucosal thickening, or thickening of the bronchial walls.

Bronchiolitis.—Inflammation of the smaller bronchi may lead to their occlusion, with multiple areas of alveolar collapse, which may give a picture very reminiscent of coarse miliary tuberculosis. The condition may be found in children suffering from measles, and is then almost invariably accompanied by enlargement of the bronchial and hilar glands. Bronchiolitis also occurs in

influenza, and after the inhalation of irritant gases (chlorine, phosgene, etc.). A classical case (quoted by *Sante*) occurred as a result of the Cleveland fire disaster. In bronchiolitis the lower lobes are predominantly affected.

The worst type of bronchiolitis on record occurred in the measles epidemic in the American encampment near Paris in 1919. It was fatal in nearly 60 per cent. of cases, and the pictures showed a generalised miliary appearance from apex to base. Similar cases were seen in an outbreak of typhus in Vienna, and the radiograms showed a streaky appearance in addition to the miliary appearance—the streaks probably being due to active hyperæmia of an extreme degree. Autopsy showed these cases to be true bronchiolitis.

Bronchiolitis obliterans usually follows gas poisoning or other acute irritative processes and leads to a permanent mottling of the lung fields, due to obstruction of the small bronchi by fibrosis. *Assmann* has described the post-mortem appearances in such a case. Radiologically, it resembled miliary tuberculosis.

Chronic Bronchitis is stated to cause changes in the bronchial walls which are visible in the X-ray, and are attributed to thickening of the mucous membrane, or to fibrous changes in the wall ("peribronchitis"). Radiographic evidence of these changes seems to the writer to be extremely unconvincing. Parallel streaks are sometimes pointed out as being due to thickened bronchial walls on either side of a bronchial lumen. In the hilar region, it is true, bronchial and tracheal walls are recognisable as parallel lines or rings, but outside this region the "parallel streak" appearance is usually attributable to superimposition of translucent bronchi upon vascular densities, and the translucent bronchus may often be seen crossing the latter obliquely. The writer feels convinced that a diagnosis of bronchitis, or of peribronchial fibrosis, on a supposed increase in density of trunk shadows is usually impossible. He also believes that in estimating this density, in any given case, insufficient consideration is paid to the question of contrast. It must be the case that the density of lung detail depends to a very large extent upon the degree of distension of the alveoli immediately surrounding. We have abundant proof of this if films are made, for example, in inspiration and expiration and then compared. Similarly, the size of the alveoli surrounding the trunks varies in different patients, as does also the volume of lung parenchyma separating the different trunks from one another. A moderate degree of emphysema of the alveoli will increase the contrast between the lung and the trunk shadows. A greater degree of emphysema, on the other hand, may reduce the contrast by increasing the scattered radiation. Such a loss of contrast is often seen in the emphysema which accompanies chronic long-standing silicosis of moderate severity; for example, in men who have worked as stonemasons during their entire lives. The hilar shadows in such a case are of less than normal density owing to the marked perihilar emphysema. This factor, varying contrast between alveolar tissue and the vascular and connective tissue strands of the

stroma, plays an important but often incalculable rôle. It makes the estimation of the actual density of the vascular and bronchial markings extremely difficult: any apparent increase in density should therefore be interpreted with great caution. The problem is further complicated by differences in exposure, which may be technical (penetration, milliamperage, and exposure time), or natural, due to the thickness of the overlying soft tissues, breast, subcutaneous fat, and so on.

It is true, of course, that a more abundant and richer network of lung markings does occur in conditions known to be associated with peribronchial and perilymphatic fibrosis (silicosis, asbestosis). It is also true that lung markings are notably increased in back pressure lung (mitral stenosis). In the former case there is predominant involvement of the more peripheral parts, where lung arborisations are visible in situations where they are normally invisible. In the latter case the markings are widened (dilatation of the vessels), and show a well-marked "diminuendo" when traced from hilum outwards. In bronchiectasis, again, there is often a very striking pattern of coarse strands radiating towards the bases, the anatomical basis of which is, quite certainly, peribronchial fibrosis and collections of secretion. Local atelectasis of the alveoli immediately surrounding the bronchi may play a part (*Assmann*). But leaving these examples on one side, the writer believes that normal variations in contrast make it almost impossible to read much of real value into the apparent density of the lung stroma as seen in radiograms, and that comment upon these points should be avoided unless a clear opinion can be given on the probable or actual cause of the appearance described.

Broncholiths

Broncholiths ("lung stones") may occur (1) from calcification in lung tissue degenerated after necrosis due to infection, or in tuberculous glands; (2) from calcification around aspirated foreign bodies (pneumoconiosis); (3) from precipitation of calcium present in excess in the blood; (4) from metastases containing bone-forming cells, (5) in bronchiectatic cavities. They consist, as a rule, of calcium phosphate. They may erode the wall of a bronchus and be coughed up. Hemoptysis, bronchial spasm, and asthmatic attacks precede the attacks. Some patients repeatedly expectorate lung stones. They are occasionally demonstrated radiologically. *Pendergrass* and *de Lorimier* record two cases, in one of which the shadow of the broncholith was seen at the apex of a triangular consolidated area of lung. After expectoration of the stone, both shadows disappeared. In a case reported by *Blecher* the stone was hidden in a pneumonic shadow.

Tuberosc formation of bone occurs in lung tissue in old cases of mitral stenosis. The radiological appearances are those of a "back pressure" lung, with engorged hila. The lung fields are studded with sharply defined rounded calcified shadows (*Salinger*).

A delicately branching bone formation in the lung tissue has also been described in elderly patients, which is not related to the bronchial walls and does not follow their course (*Pneumopathia osteoplastica racemosa*. *Simmonds*.)

Calcium metastases may be found in the lung tissues in cases of osteitis fibrosa with rapid osteoporosis of the skeleton.

BRONCHIECTASIS

Subdivisions of bronchiectasis are numerous, according to the viewpoint adopted. It may be saccular or cylindrical; diffuse or localised; spindle-shaped or, if a series of sacs are present, varicose; acute or chronic; wet or dry; congenital or acquired. Perhaps the most useful division from a radiological standpoint is that into congenital and acquired forms, but even here there is no clear-cut radiological distinction: nor are the ætiological factors so thoroughly understood that we can assign a given case unhesitatingly to one or the other group.

Congenital bronchiectasis is often associated with other malformations of the bony thorax, ribs, or lung, and, like these, may be familial (*Kerley*). There are two forms: (a) Cylindrical enlargement with little inflammation of the bronchi, often symptomless and often limited to one lobe. There is failure of development of the alveolar sacs with enlargement of the bronchi. It is often discovered accidentally. (b) Cystic. The alveoli show complete failure of development and the bronchi grow out as a thin-walled cyst which may replace an entire lobe or lung. This ballooned, imperfectly developed bronchus is lined with cylindrical epithelium. It is clear that this ætiology is essentially exactly the same as that of congenital cystic lung—namely an agenesis, or failure to develop of the terminal elements of the bronchial tree. This development normally continues for several years after birth and in some cases up to puberty. Cases of congenital bronchiectasis may be met with in the foetus, the new-born, or at any period later in life. Congenital bronchiectasis and congenital cystic lung would therefore appear to be one and the same condition.

Congenital bronchiectasis may occur anywhere in the lungs. The saccular and cystic form is the more common, and shows numerous circular shadows, varying in size from a quarter of an inch to an inch in diameter. Secretion is sometimes visible in a few of them, but is often absent; and clinically this form tends to be without profuse expectoration and may be entirely dry. If it is extensive, there is concomitant retraction of the lung, falling-in of the chest wall, and mediastinal displacement. The sacs may become infected at any time. Lipiodol enters the cavities freely. The bronchi leading to them are not as a rule dilated. The cylindrical type of congenital bronchiectasis exactly resembles the acquired form.

Acquired bronchiectasis usually originates in an acute respiratory infection. This may lead to ulceration, destruction of muscle and elastic tissue of the bronchial wall, and replacement by granulation tissue and fibrous tissue. At first



FIG 118 —Bronchiectasis Bullous emphysema at right base

cal, it may later become saccular. In some cases chronic bronchitis may bronchiectasis. Whether a primary fibrosis surrounding the bronchi may their dilatation is doubtful. Fibrosis is often present, but probably very. Bronchiectasis not infrequently complicates tuberculosis, the

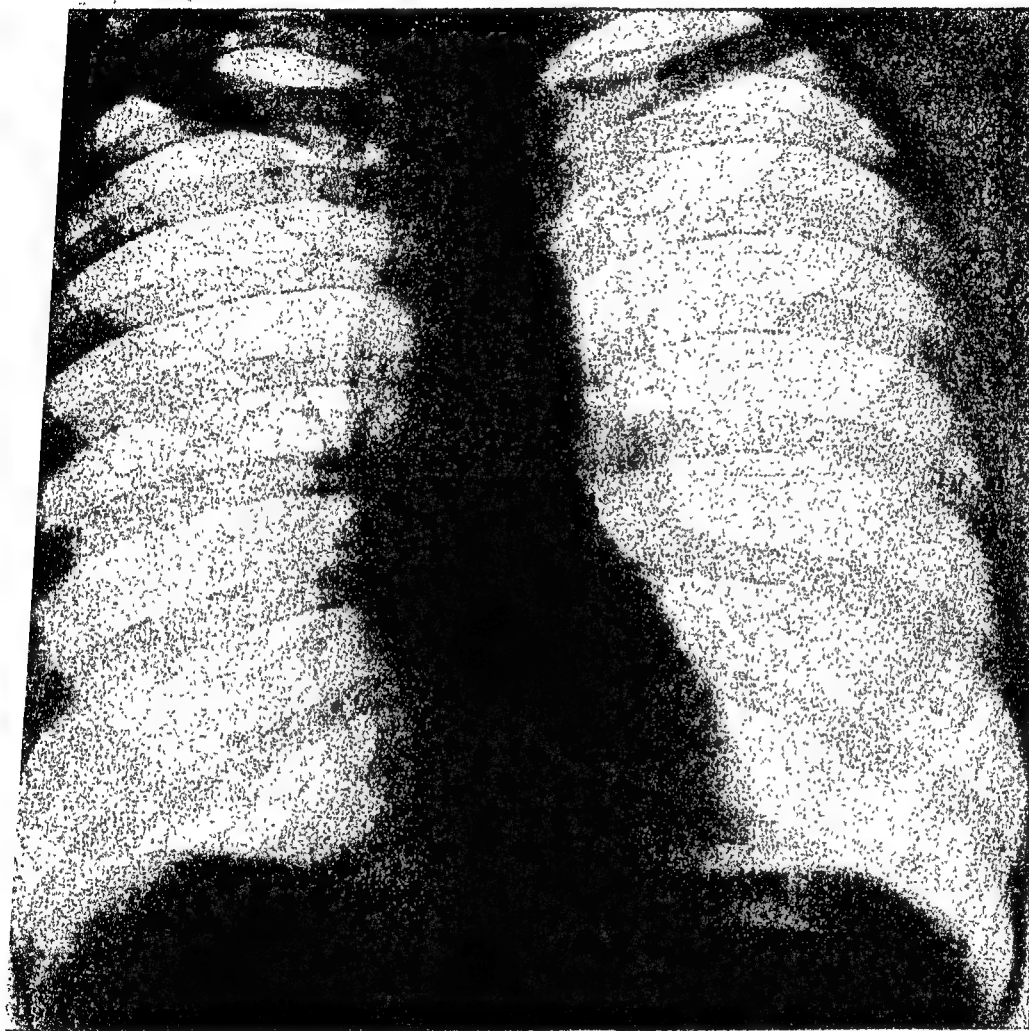


FIG. 119.—Congenital bronchiectasis. Note slight “honeycomb” appearance in lower part of right upper lobe, confirmed by lipiodol injection, see Figs. 120, 121.

bronchi leading to the affected region being first damaged by ulceration of their walls, and subsequently becoming dilated.

Atelectatic Bronchiectasis.—If a lobe becomes atelectatic as a result of bronchostenosis from any cause, bronchiectasis is likely to occur in a short time unless the stenosis is relieved. Probably the majority of “atelectatic bronchiectases” in a lower lobe are of the acquired type, and the sequence is collapse,

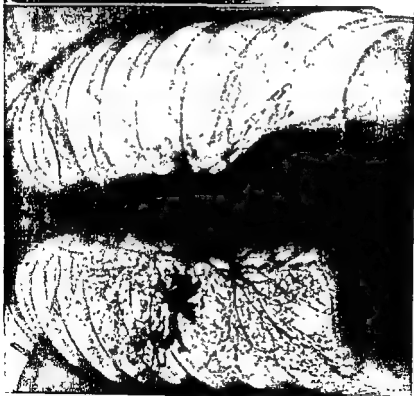


FIG 120—Congenital bronchiectasis

F 20 History of repeated haemoptyses for many years
No pyrexia, slight cough, blood sedimentation rate normal



FIG 121.—Congenital bronchiectasis Lateral view.
treated as pulmonary tuberculosis, although sputum never positive.

infection, bronchiectasis—not bronchiectasis—followed by collapse. *Ellis* (1933), reporting 5 cases of triangular basal shadow in children, agrees with this view. In each case bronchopneumonia had occurred in early childhood. The children, aged 8–13, had mild cough and scanty sputum. The triangular shadow was proved by lipiodol in each case to contain dilated lower lobe bronchi. Atelectasis of an accessory lobe, emphasised by *Kerley*, is probably a relatively uncommon cause of the basal triangular shadow. Minor degrees of bronchiectasis, with or without collapse, may be recovered from, and cases are on record of restoration to normal of bronchi which have shown, when outlined with lipiodol, very definite cylindrical dilatation.



FIG. 122.—Atelectatic bronchiectasis, right lower lobe. The lower lobe is shrunken to a triangular mottled shadow. Male, 17. Disease dates from pneumonia at age of 1½ years.

Radiological Appearances

Bronchiectasis in a plain film shows coarse mottling usually towards the bases and filling up the triangle between the heart and diaphragm. The basal striation is increased; this is due to the thickening of the bronchial walls, which become visible. They cease, as a rule, at the level of the diaphragm and do not continue downwards behind the dome as do the vascular shadows, and, unlike vascular shadows, do not show regular diminution in size or regular branchings. The cavities are often difficult to make out, but tend to lie along the translucent streaks of the larger bronchi. Occasionally small fluid levels may be seen. Fibrosis and atelectasis frequently accompany the condition. As a result of this, the lung markings of the lower lobe tend to be grouped into the angle between the heart and diaphragm and the branches of the pulmonary vessels to the lower lobes may show a concave type of radiation instead of

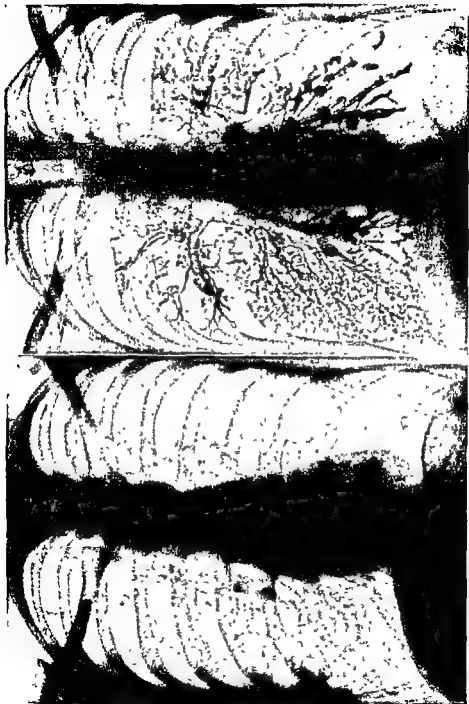


FIG. 123.—Bronchiectasis and emphysema. Except for a few faintly indicated cavities in the left lower hilar region, there is nothing to suggest bronchiectasis. But note suspicious localized emphysema at left base.

FIG. 124.—Same case as Fig. 123. Lipiodol shows varicose bronchiectasis in both lower lobes.

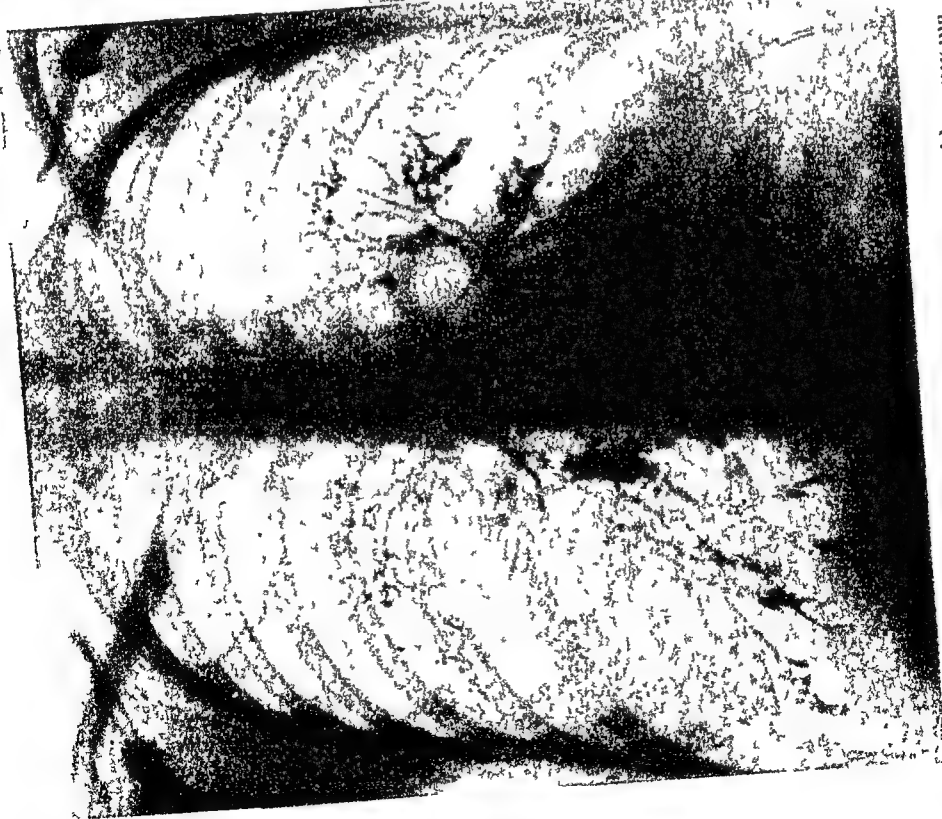


FIG. 125.—Bronchiectasis, left lower lobe, following old empyema.
Note resected rib and fibrosis of lower lobe

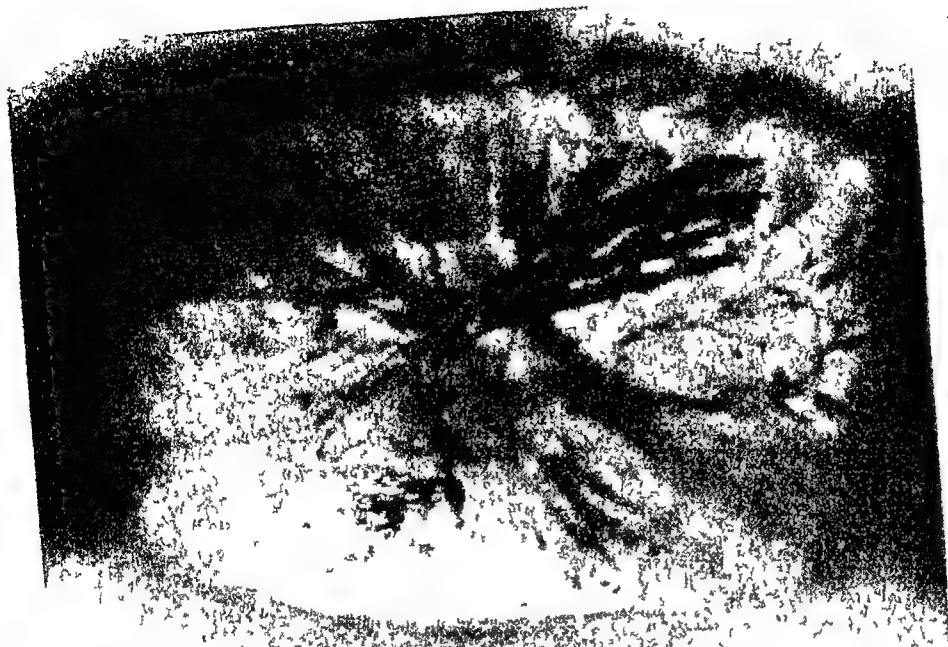


FIG. 126.—Lateral view to Fig. 125. Cylindrical bronchus-ectasis of "glove finger" appearance

springing boldly out into the lung fields. If the atelectasis is marked, the lower lobe shows as a dense triangle in the region just mentioned and on the left side often, and occasionally on the right, may be completely hidden by the heart shadow in the postero-anterior view. In the lateral view the characteristic atelectatic posterior triangle is found with backward shift of the main interlobe, and may be "honeycombed" with cavities.

It frequently happens that there is an intense emphysema on the bronchiectatic side masking the bronchiectasis or at the most allowing only a few rounded cavities to be faintly seen through it. This emphysema being as a rule in the middle and upper lobes, it is important to remember that the costo-phrenic angles may be well filled and apparently normal even in the presence of a lower lobe collapse as a result of this emphysema of the upper and middle lobes. The lung markings then radiate from the upper and middle part of the hilum and the lower lobe collapse is betrayed by the absence of the normal lower lobe markings descending behind to the diaphragm in the postero-anterior view. Lipiodol should be used in such a case.

Neither the clinical nor the radiographic features of bronchiectasis are invariably clear-cut. There are cases of bronchiectasis with well-marked physical signs, and insignificant or absent radiographic signs. There are others with almost entire absence of physical signs, and no purulent expectoration or severe *tæxæmia*. *Kerley* has stated that this occurs in one-half of the cases in children. The writer's experience is that, though it is often possible to suspect bronchiectasis from the condition of the lower root shadows, the clinical signs are more reliable than the radiographic film, unless lipiodol is used. Saccular bronchiectasis and large isolated bronchiectatic cavities containing secretion may be very obvious. So may upper lobe bronchiectasis, resulting from chronic tuberculosis. Here the dilated bronchi become very obvious by contrast with the surrounding indurated lung.

If a suppurative inflammatory condition develops in the surrounding lung, a homogeneous shadow develops, in which ragged cavities may appear (bronchiectatic abscess).

Kerley has contributed the following observation :

"I have seen several cases of bronchiectasis in which the whole of the affected lobe suddenly became opaque. After two or three days' postural drainage the opacity disappeared and the condition was as before; it was apparently due to a transient pneumonitis. In such cases it is safer not to diagnose a bronchiectatic abscess unless cavitation is visible, or until postural drainage has been tried for a few days."

It should be remembered that hæmoptysis may be the only important symptom and that bronchiectasis should always be considered in cases which do not show any other explanation, e.g. tuberculosis or neoplasm, for the symptoms. Hæmoptysis may be the only striking symptom in the dry form (*Forme sèche hæmoptoïque*) of the disease. As a rule lipiodol injection

will establish the diagnosis and this procedure alone can be relied upon to define the position and extent of the bronchial dilatations, and to determine whether the disease is unilateral or bilateral.

Lipiodol Injection

TECHNIQUE.—(1) *The transglottic method* is usually employed. The pharynx is first sprayed with 4 per cent. cocaine. The larynx and upper trachea are also well sprayed with a weaker solution, which is dropped through the vocal cords. When the cough reflex is abolished a curved catheter is passed over the back of the tongue, and lipiodol, warmed to body heat, is injected into the larynx, about 1 c.c. at a time, at the end of each quiet inspiration. If only the lower lobes are to be injected the patient sits up. Inclination towards one side will fill that lower lobe better. Further tilting to the side will fill the lower parts of the upper lobe, but for good filling of the latter a recumbent position, with the patient on one or the other side, is usually necessary. 20 c.c. (10 into either side) will usually succeed in outlining both lungs at one sitting if so desired. Fluoroscopic control of the injection, with films made as soon as the oil enters the small bronchi, is desirable. Flooding of the alveoli tends to obscure detail. This, however, rarely occurs in the affected area in a case of bronchiectasis, since the alveoli do not fill at all and the bronchi terminate blindly. Lateral views are essential, and oblique views are often necessary in addition, since the right and left lungs are superimposed in the full lateral view.

(2) *The Nasal Catheter Method*.—A number 9 gum elastic catheter, or in children a smaller soft rubber catheter, is passed into the trachea after thorough cocainisation of the nasal passages, nasopharynx, and larynx. The latter is preferably done by dropping 1 c.c. of 2 per cent. cocaine through the cords, controlled by indirect laryngoscopy. The catheter is passed, after coughing has subsided, through the nose until it touches the larynx. The patient inspires deeply and the catheter is pushed into the trachea. If cough supervenes, another cubic centimetre of cocaine solution is injected through the catheter. The catheter is left in situ, fixed by a plug of gauze in the nose. The end of the catheter should be just above the tracheal bifurcation; the lipiodol, warmed to body temperature, is injected. The position of the patient is as follows:

(1) For the lower lobe—sitting, inclined to that side.

(2) For the upper lobe—lying upon side, alternating with prone position. Some tilt, head end downwards, is permissible, but the head itself must be kept raised. Fluoroscopic control is essential.

(3) *Direct Injection into the Trachea through the Crico-thyroid Membrane*.—For this the patient lies supine on the X-ray couch, and the skin is anæsthetised. The same hypodermic is used to inject 1 c.c. of cocaine through the crico-thyroid membrane into the trachea. A curved needle with lipiodol syringe attached is then inserted into the trachea and the injection is made. This was the original

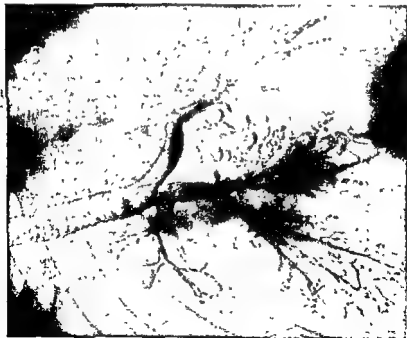


FIG. 127.—Varicose bronchiectasis, becoming sacular.
Left anterior oblique view.



FIG. 128.—Bronchus (asis, sacular type.

method and is still sometimes used. It may be difficult to keep the needle in the trachea, especially if there is much cough, and it is not uncommon to find cases in which the injection has obviously penetrated into the paratracheal tissues with unsightly, though apparently not dangerous, results.

Appearances of Bronchiectatic Cavities outlined by Lipiodol

CYLINDRICAL BRONCHIECTASIS.—The hilar shadow is, as a rule, increased owing to peribronchitis and enlargement of the glands. The dilated bronchi are shown as dense finger-like shadows; if the bronchi are completely filled with lipiodol. If they are incompletely filled, the bronchi are shown as translucent streaks with parallel dense walls to which the lipiodol adheres. End-on bronchi appear as rings or, if filled, as opaque spots. The terminations of the bronchi do not show the usual fine branching. A few club-shaped irregular finger-like processes take their place, and the finer bronchi and alveoli fail to fill. The affected bronchi fill passively and remain filled for a considerable time.



FIG. 129.—Saccular bronchiectasis.

SACCULAR BRONCHIECTASIS.—The cavities show rounded, circumscribed shadows.

In the erect position a lipiodol pool with fluid level is seen in the lower part of each cavity. If a cavity is full of secretion, the lipiodol does not readily mix with it, but may surround it as a ring shadow with clear centre, reminding one of a diverticulum of the colon containing faeces, when outlined with barium. The cavities vary in size. Sometimes a bunch of small grape-like shadows occurs, grouped round

the smaller bronchioles. Very small cavities occur in children after whooping-cough (bronchioectasis) (*Sicard, Forestier, Schinz*) and show a somewhat widespread distribution.

VARICOSE BRONCHIECTASIS is an intermediate form between the cylindrical and saccular types, affecting the large and medium-sized bronchi. The bronchi are tortuous, and show along their course groups of smallish cavities sometimes arranged in linear fashion, so that they resemble a string of pearls. The condition is often unilateral.

Lipiodol rarely fails to enter bronchiectatic cavities, whether acquired or congenital. It usually enters congenital cysts. It frequently fails to enter abscess cavities, cavernous neoplasms, and tuberculous cavities, because the bronchus leading thereto is blocked by secretion, slough, pus, or granulation tissues; it never enters emphysematous blebs and bullæ.

BENIGN TUMOURS OF THE BRONCHI

Benign tumours of the bronchi (adenoma, fibroma, lipoma, myoma, chondroma, or polypus) are not so rare as the literature would suggest. *Morlok* and *Pinchin* found nine in 150 bronchoscopic examinations in the course of four and a half years. Though benign, they may produce serious secondary changes in the lung, as a result of bronchial occlusion. A benign tumour should also be borne in mind as a possible cause of hæmoptysis in suspected cases of tuberculosis with negative or atypical findings. Hæmoptysis may be the only symptom for many years.

When the growth is large, atelectasis may occur suddenly. Usually obstruction is slow and gradual, and pulmonary suppuration occurs insidiously. Bronchiectasis is a common sequel. Empyema may occur.

A case of polypus, causing complete atelectasis of a lower lobe, is illustrated in Fig 130. The polypus, about the size of a large pea, was removed by *Graham Bryce*. The atelectasis cleared up rapidly, and the lung was entirely normal on re-examination a few weeks later. The patient remains well.

CONGENITAL CYSTS OF THE LUNG

Congenital cystic malformations of the lungs were first described by *Fontanus* in 1638 as a result of post-mortem findings in a child of 3 months, who died during an attack of cyanosis. The lungs were replaced by large air-containing cavities communicating with the bronchi. Of recent years numerous cases have been reported with similar post-mortem findings. *Koontz*, reviewing the subject in 1925, collected 108 cases. Since then others have been reported in the pathological and medical literature, and many cases have also appeared in the radiological literature. *Schenk* in 1936 collected 232 cases and described the radiological appearances. The congenital origin of the disease is fully established by its discovery in the fœtus and in still-born infants, or in

children dying in very early infancy. *De Lange* describes an interesting case in a child who died in a cyanotic attack when 7 weeks old. The first of a series of attacks had occurred at the age of 7 days. Autopsy showed numerous cavities and bullæ in the left lung, in places reaching the surface. Some of the cavities did not obviously communicate with one another, or with bronchi. The cavities were lined with cylindrical or cuboidal epithelium. *De Lange's* case is closely paralleled by that of *Argyll Robinson*.

From a radiological standpoint the principal interest attaches to those cases in which the disease is latent, and is discovered, often as a chance finding, in

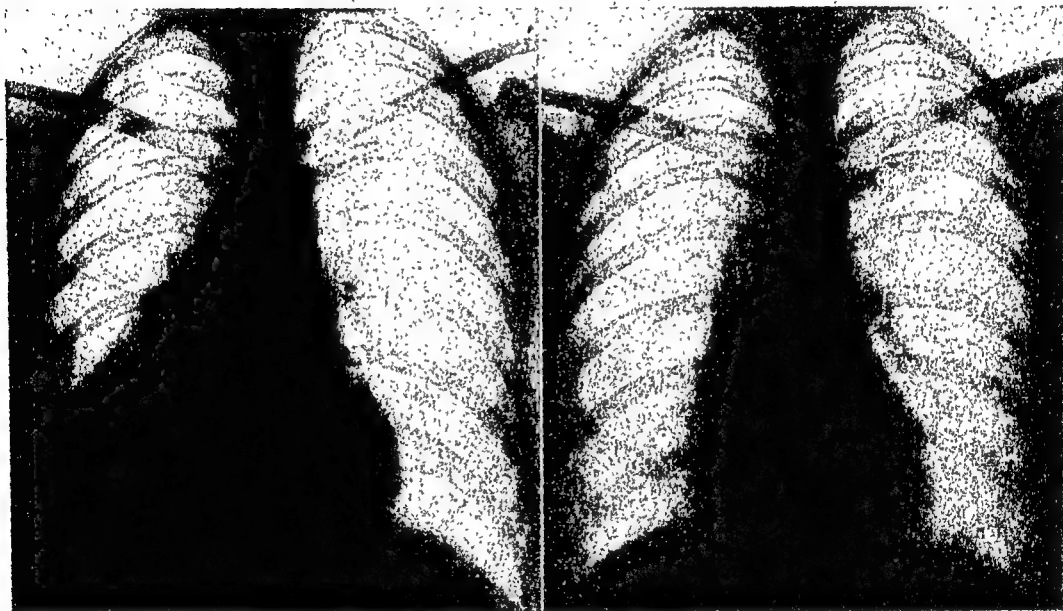


FIG. 130.—Papilloma of the right lower lobe bronchus. Atelectasis. Pea-sized adenomatous polyp removed by bronchoscopy. Same case three months after operation.

older children or adults. Such cases are not rare, the writer having observed six in the last two years. About half the cases are first recognised after the fifteenth year. A further question of considerable interest is the relationship between bronchiectasis and congenital cystic disease. The question has been raised by some authors whether many cases of bronchiectasis are not actually of congenital origin, representing one form of congenital cystic disease. *Kerley* has pointed out that in a review of sixty cases of bronchiectasis in childhood studied by him, one-half of the cases had no typical symptoms of bronchiectasis (i.e. no persistent cough, purulent sputum, or toxæmia), and that more than half had associated congenital abnormalities of the thorax. Two of the patients were brother and sister. He commented upon the not uncommon

association of bronchiectasis and atelectasis of a normal lobe, or of an infracardiac accessory lobe. The familial incidence and the occurrence in an atelectatic infracardiac lobe have been noted also by other observers (*Richards*).

Ætiology

Various authorities disagree as to the mode of origin of air cysts. *De Lange* divides them into: (1) Cases of foetal inflammatory bronchiectasis. (2) Cyst



FIG. 131 —Thin-walled congenital cyst in right lower lobe.

formation in hyperplastic lung tissue. ("Foetal Adenoma." *Stoerk*.) (3) Agenetic bronchiectasis. (4) Lymphangiectasis.

By agenetic bronchiectasis is meant a failure of the terminal buddings of the bronchial tree to develop into alveoli. This leaves spaces which, with the further growth of the thorax and lungs, can only be filled by dilatation of the unsupported bronchi. In post-natal life these originally small bronchiectases are increased in size by valvular action and over-inflation. No one theory

would appear satisfactory to explain all the recorded types.

Types of Congenital Cystic Lung

Grawitz divided the lesions into two groups: (1) *Bronchiectasis universalis*, in which the whole or part of a lobe, or a whole lung, may be occupied by miliary cysts distributed through normal parenchyma, or converted into large multilocular or single sacs; and (2) *Bronchiectasis telangiectatica*, a circumscribed cystic enlargement of part of a bronchus, with or without communication with other bronchi. The first group constitute the "multiple type," the second the

"solitary air cyst." The cysts are derived from bronchial tissue and contain muscle, fibrous tissue, and cartilage; they are lined with epithelium, sometimes ciliated, and may contain papillary ingrowths. Clinically many cases are silent, but there may be repeated attacks of bronchitis, with cough

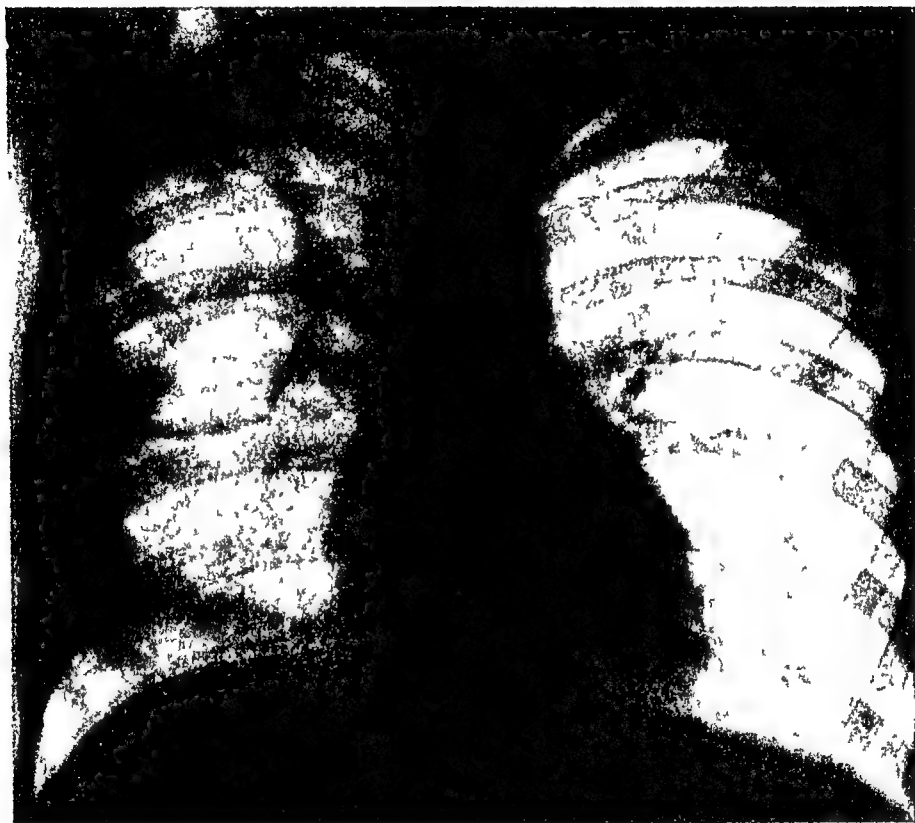


FIG. 132.—Congenital cystic lung, verified at operation.

and expectoration, and elevation of the temperature. In young children, in whom solitary cysts are more common, alarming attacks of cyanosis may occur.

Radiological Appearances

These are very variable, and depend upon the size and distribution of the cysts, which may be found in the child or in the adult. One-third of all cases occurred in adults in *Koontz's* series; in one-quarter the disease is bilateral.

(1) **SOLITARY AIR CYST.**—A rounded air-filled shadow, often in the hilum region, or at one base, showing no evidence of inflammatory reaction in the surrounding lung. The wall is sharply defined and usually thin. A fluid level may be present. The orifice of communication with the bronchus is often so small that lipiodol cannot be introduced into the cavity.

(2) **GIANT AIR CYST**, simulating pneumothorax. This is an extremely important though rare type, in which, in extreme instances, the entire hemithorax may be translucent, and marked displacement of the heart and mediastinum may be present. Symptoms may be entirely absent. The lung markings may be missing everywhere, or present in some circumscribed portion of the lung. Interesting cases of this type have been published by *Freedman, Crosswell*

and *King, Jacobaeus, Ruckensteiner* and *Hortnagl*. The writer has observed one similar case.

In *Ruckensteiner* and *Hortnagl's* case, X-ray examination during exploration by paracentesis showed an interesting finding. The needle, on entering the pleural cavity, pushed the cyst wall (formed by visceral pleura) away from the parietes. At this moment negative pressure was recorded, proving that the needle lay in the pleural cavity. Next, the needle penetrated the cyst wall, and positive pressure was recorded, proving that the needle was then in the lung—which consisted of one enormous balloon cyst.



FIG 133.—Cystic lung • left lower lobe is occupied by large thin walled cavities, and diminished in volume.

(3) **MULTILOCULAR CYSTS** show the lung to be honeycombed in the affected part with oval or circular translucent cavities, with or without fluid levels. They are sometimes hexagonal from mutual pressure. The partitions are thin and sharply defined, and there is no reactive inflammation around them unless infection has occurred (*Kuhlmann*). This is an important point of distinction from tuberculous cavities. There is no fibrotic drag, and therefore no displacement of the heart and mediastinum. Lipiodol may sometimes enter the cysts, but will not always do so.

A type of cyst occurring in the upper lobe, in which the appearance at first suggests a bullous emphysema, has been noted in one case by the author, and

is similar to cases described by *Flemming Møller*, by *Freedman*, and by *Dethmers*. The abnormally translucent areas in the upper lobe were separated by thin strands radiating out to the periphery; where these reached the pleural surface they terminated in small peaks or cusps. The cysts contained no fluid and lipiodol could not be made to enter them. In *Dethmers*' and *Cherry*'s cases a beautiful outlining of all the cysts with lipiodol was obtained.

Minute disseminated cysts may stimulate the radiological appearances of disseminated tuberculous disease, or of a disseminated mycotic infection.

A case reported by *Kerley* is possibly an example of this type of the disease.

(4) FLUID CYSTS.—In rare instances a congenital cyst is entirely filled with fluid and shows a circular or oval opacity. This may occur with lymphangiectatic cysts or in those of bronchial origin. They are usually solitary and grow very slowly. Rupture into a bronchus may occur.

Kirklin shows an example of a fluid-filled cyst of bronchial origin removed by *Harrington*. Its shadow was well defined, rounded and homogeneous, and fused medially with the mediastinum. It was indistinguishable from any other benign tumour.

Differential Diagnosis.—Single air-containing solitary cysts are distinguished from other cavities only by their delicate circular wall, without any surrounding inflammatory thickening. A trace of fluid may be present at the bottom of the cavity. If the cyst contains more fluid, and especially if inflammatory changes have followed rupture into a bronchus, abscess or hydatid cyst may be simulated.

Large or giant air cysts are usually single, though occasionally two or three are found together. They are demarcated from the normal lung by a thin rounded line, and lung markings are absent within them. The difficulty with the very large cysts is to distinguish them from pneumothorax, for the thin outer wall when in contact with the chest wall is invisible, and the mediastinum may be displaced across the midline. Differentiation depends upon the absence of a clearly defined collapsed lung. A diagnostic pneumothorax will often differentiate the cyst clearly by showing its outer wall. Moreover, the cyst may collapse less than the rest of the lung, and so stand out more clearly.

Multiple air-filled cysts lying close together give to the affected region of the lung a marked translucency which may be diagnosed as emphysema, but the fine septa form a network of crossing strands or cobwebby shadows which have a quite different appearance from the normal vascular markings which in emphysema can be traced back to the hilum and show regular branchings.

Emphysematous blebs and bullæ lie on the surface of the lung; cysts lie in its substance, though they may extend in places to the pleural surface, which is finely notched where the septa meet it.

In acquired bronchiectasis there are many cases in which a differentiation cannot be made. Acquired bronchiectasis is more often basal, and more often accompanied by fibrosis. The usually long-standing infection causes much

thickening of the walls of the cavities. The size of the bronchiectatic cavities tends to increase peripherally. Vascular markings are often increased. Gross fibrosis and atelectasis are probably more common in the acquired form, but atelectasis may coexist with either.

Peirce and Dirkse have shown that X-ray appearances like those of congenital cysts may result from lung infection. It is well known that vesicular



FIG. 134.—Congenital cystic lung, left lower lobe. Verified by operation

emphysema may appear during the course of pneumonia and disappear gradually or rapidly. *Peirce and Dirkse* have found examples of its persistence after acute respiratory infections in the form of single or multilocular cyst-like areas. In their six cases, five of which were in children under 13, a congenital origin could be excluded on the evidence of previous radiograms, or, in some cases, by histological investigation after lobectomy. These workers suggest that congenital cystic lung is probably less common than is at present believed

and that many cases of supposed congenital cysts are in fact acquired. They suggest that "cysts" of the lung fall into the following groups :

(1) True congenital cysts, which, on the evidence of post-mortem statistics in children, are rare.

(2) Alveolar emphysema, in chronic interstitial pneumonia, and bullous emphysema, in cases of expiratory obstruction. e.g. asthma. Fluid levels are absent.

(3) Cystic bronchiectasis, usually resulting from acute respiratory infection (bronchopneumonia in childhood), which accounts for most cases of honey-comb lung. Fluid levels are often present.

, (4) Pulmonary pneumatocele (alveolar or lobular ectasia). They have demonstrated in two cases the origin of a thin-walled cyst, which may be of the giant type, in the course of a pulmonary infection and its persistence for more than a year with subsequent disappearance.

Some cases are suitable for surgery. Lobectomy has been successful in a high percentage of cases (*Sauerbruch*).

CHAPTER XX

ATELECTASIS

"ATELECTASIS" and "collapse" are used as synonymous terms in the radiological literature to signify collapse of the lung alveoli. It may be congenital or acquired.

Ætiology and Pathology

CONGENITAL ATELECTASIS results from failure of the lungs to expand at birth, the infant rarely survives. In infants, in films taken at the end of expiration, the lungs may appear so opaque that atelectasis is simulated.

A case of congenital atelectasis in a new-born child has been recorded by *Kerley*. A radiogram taken six hours after delivery showed complete opacity of both lungs. The infant was deeply cyanosed and died within a short time. Post-mortem showed complete atelectasis of the left lung, and only traces of air in the right. There was no abnormality of the bronchial tree.

Complete atelectasis of both lungs in an infant has been observed by *Mather*. The cause was tracheal obstruction by an enlarged mediastinal gland which pressed upon it from behind above the bifurcation. The radiogram of this case during life, and a post-mortem radiogram of the lungs injected with lipiodol, are shown in Figs. 135, 136.

ACQUIRED ATELECTASIS results from bronchial occlusion. The cause may be :

(a) Endobronchial: e.g. neoplasm, foreign body, mucus, blood, granulation tissue, pus.

(b) External; due to pressure of tumour, enlarged mediastinal glands, aneurysm, etc.

If a large bronchus is obstructed, massive collapse occurs. If a small bronchus is obstructed, the area involved is correspondingly small, and may consist only of a lobule, or of a part of a lobule. The condition may be present in association with many forms of lung disease, and the rapid appearance and disappearance of lung shadows so often found, especially in radiograms of children, is, in many cases, due in whole or part to this factor. The aphorism "think atelectasis" is sound doctrine.

Atelectasis of an entire lung results from the blocking of a main bronchus. It is fairly common in bronchial carcinoma, and after the inhalation of foreign bodies. Soft vegetable foreign bodies, such as peanuts, or friable material of any kind, are particularly dangerous.

Post-operative massive collapse affords a clear-cut example. This condition,

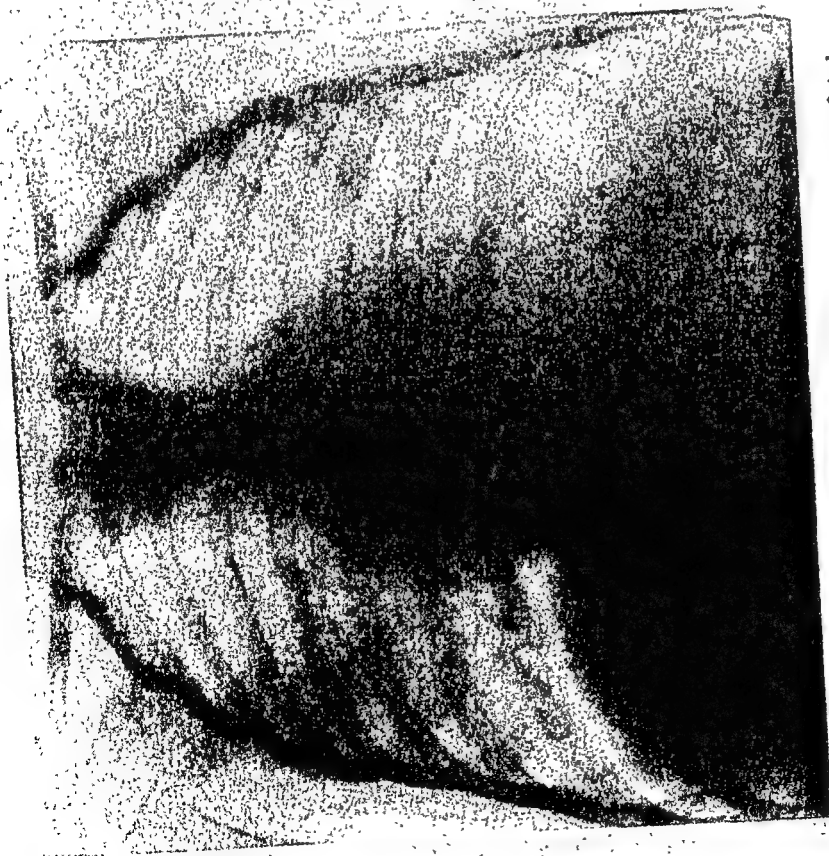


FIG. 135.—Atelectasis of both lungs due to pressure of enlarged tuberculous glands in a child of 2½ years old.



FIG. 136.—Autopsy specimen of the same case as Fig. 135 injected with lipiodol. The gland, seen as a round shadow above the eparterial bronchus, had completely obstructed the trachea.

made known to us by *Pasteur* (1908), *Bradford*, *Elliott*, and *Dingley*, has been studied radiologically by *Bowen*, *Sante*, and *Boland* and *Sheret*, among others. Its post-operative incidence is diminishing with the use of barbiturates for anaesthesia, since these stimulate the cough reflex. The writer had an opportunity of observing the work of *Boland* and *Sheret* at Ancoats Hospital. They examined, with a portable apparatus, 261 patients after abdominal operations;

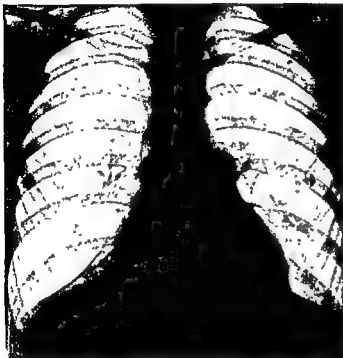


FIG. 137.—Bilateral collapse of lower lobes. The triangle of the left lower lobe is just visible through the heart shadow. Marked compensatory emphysema fills the costo phrenic angles.

the anaesthetic used was usually ether. Six of these had a classical collapse of the whole of one lung or of one lower lobe, with clinical evidence of cardiac displacement. The examination brought to light thirty further cases, without clinical cardiac displacement. (Eleven bilateral, ten in left lower lobe, nine right lower lobe.) It was found most frequently after upper abdominal operations.

The theories of *Pasteur*, *Bradford*, *Briscoe*, *Scrimger*, *Scott*, *Sante*, and others invoke a neuromuscular reflex affecting diaphragm, chest wall, or bronchioles; reflex inhibition of diaphragm movement is probably a contributory cause, but the main factor is blocking of a bronchial lumen by secretions.

When the obstruction is removed by rolling the patient on to the unaffected side and making him cough, mucus is dislodged, and the atelectasis rapidly clears up (*Sante's* manoeuvre). The occurrence of the condition, in the series referred to, was prevented by nursing with the bed foot raised and the head kept low for twenty-four hours following operation.

Clinical Features

During the first twenty-four hours there is a steady rise in pulse, respiration rate, and temperature, with shallow respirations and restricted

mobility of the chest. Percussion shows diminished resonance and breath sounds are weakened. Coarse râles are heard everywhere.

During the next twenty-four hours cyanosis remains, and the temperature and pulse are nearing their maximum. Physical signs are those of a "dead lung." On the fourth to fifth day temperature and pulse subside and the cough becomes productive, and the lung becomes normal unless inflammatory pneumonia or other severe inflammatory changes supervene (secondary bronchopneumonia, abscess). The mortality in uncomplicated cases is low.

Radiological Appearances

ACUTE MASSIVE COLLAPSE.—The region occupied by a large bronchus during the first twenty-four hours shows increased density with a mottled appearance. The mottlings are about pea size, but not very regular in size. They result from the intermingling of lobules already airless, with others in which absorption of the air is incomplete. At the end of forty-eight hours the opacity in this affected area is uniform. The oxygen is rapidly absorbed, the nitrogen not fully absorbed for twenty-four hours.

Diminished lung volume is expressed in various ways. There is diminution in size of the affected hemithorax. The ribs slope more steeply on the affected side; the intercostal spaces are narrowed, the diaphragm elevated, and the mediastinum displaced towards the affected side. The latter sign is common if the whole lung is atelectatic, but not at all common if the lower lobe only is affected. It is found in the latter case that the tendency is for the heart to be displaced backwards, but not to one side, as shown by study in the lateral view. The lower lobe is then found to be crowded into a small triangular area in the posterior costophrenic angle by the other lobes, and bounded anteriorly by a clear-cut anterior margin. The hilum is displaced backwards also. The antero-posterior view shows a characteristic triangular shadow beside the heart, or on the left side overlapped by it; broader below than above; of uniform density and bounded on the outer side by a well-defined margin, often concave towards the outer side.

The diaphragmatic elevation is, in the lower lobe involvement, most marked at the posterior cul-de-sac. In other cases there is found also an elevation of the medial part on the affected side, running up to become continuous with the heart

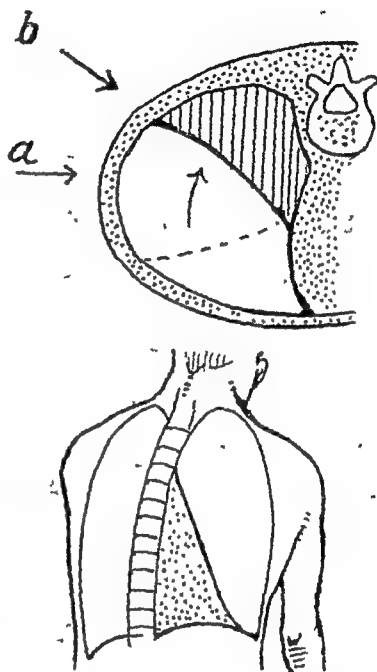


FIG. 138.—Diagram: A collapsed lower lobe may be invisible in the full lateral view (a), because its anterior surface has rotated backwards. It will show a well-defined edge if viewed from (b) posterior oblique (see Fig. 139).



FIG. 139.—Collapse of left lower lobe. The patient was rotated into the left posterior oblique position to show the well-defined edge of the lobe, not visible in the postero-anterior or full lateral positions (see text).



FIG. 140.—Melocytic middle lobe. The differentiation from interlobar pleurisy is extremely difficult: the pattern of the vascular shadows and the absence of recognizable vessels to the middle lobe support the diagnosis.



Lateral view. The middle lobe is shrunken to a narrow band in which 'lipiodol' is seen. There is marked compensatory emphysema of the upper and lower lobes.

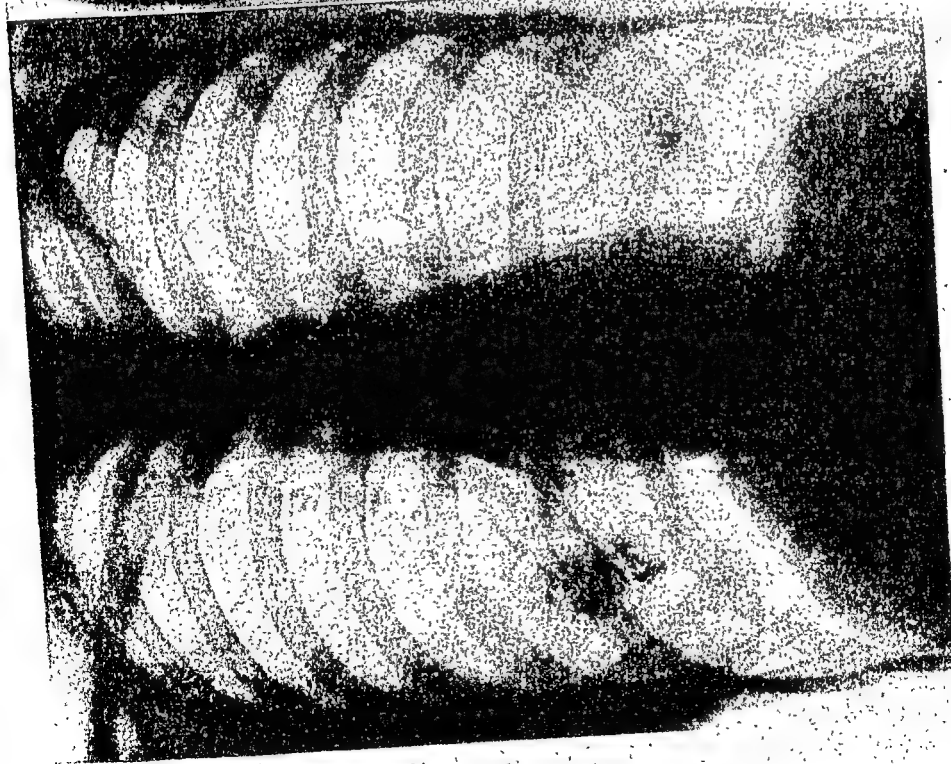


FIG. 141.—Lipiodol in collapsed middle lobe.

and filling in the cardio-phrenic angle with a triangular shadow. A similar appearance is often found in bronchial carcinoma with atelectasis of a lobe or lobes.

After re-aeration, the lung does not at once return to its normal size, being sometimes maintained in a partly shrunken condition by pleural adhesions, or by the compensatory emphysema of the other lobes. Residual local consolidation is found in a few cases.

Pulmonary atelectasis occurring in association with inhaled foreign body, neoplasm, tuberculosis, lobar pneumonia, and bronchiectasis is described under the appropriate sections. Localised areas of atelectasis due to pressure of the enlarged ends of the ribs in rickets have been described. Atelectasis from compression of the lung occurs in pleural effusions, general or localised, pneumothorax, tumours of the pleura or chest wall. Deficient aeration, amounting to partial atelectasis, also occurs in seriously ill bedridden patients, in whom diaphragmatic movement is greatly restricted. The basal parts of the lung then show an opacity which may be wrongly interpreted as bronchopneumonia or "hypostatic" congestion, but is really due to temporary lobular atelectasis.

COLLATERAL RESPIRATION.—As a result of experiments carried out by them, both in excised lungs and in anæsthetised dogs, *Van Allen* and *Lindslog* conclude that there are minute communications, as yet unidentified, between the alveoli of different lobules. According to these workers, "collateral respiration" takes place through these channels, and plays a large part in preventing the onset of atelectasis and in providing a mechanism by which the smaller bronchi, when obstructed by secretions, may be cleared: for the air in this case can seep into the alveoli supplied by these bronchi, through these collateral channels, from the adjacent normal parenchyma. The theory is somewhat revolutionary, though some of the experiments are convincing. The experiments, however, show that when a large lobar bronchus is obstructed, no collateral respiration occurs. This suggests a possible argument against the theory; for in many lungs the interlobar fissures are so shallow that large surfaces or parenchyma supplied by different lobar bronchi are actually in contact. In such a case, collateral respiration should occur from one lobe to another.

Adams and *Voricaid* found that collateral respiration did occur from lobe to lobe in some dogs, apparently as a result of physiological anastomosis with an adjacent lung lobe, and they query the statement of *Van Allen* that it only occurs in occlusion of the smaller bronchi.

The work is of great interest, but needs confirmation. The writer believes that the conclusions of *Rabin* that segmental atelectasis can only occur in an inflamed, and not in a healthy, lung are not yet justified. The writer has, at all events, observed its occurrence after sudden obstruction of a medium-sized bronchus by foreign body or hæmoptysis.

LINEAR ATELECTASIS (*horizontal linear shadows at the lung bases*).—Very puzzling transverse lines are sometimes seen at one lung base, right or left, crossing the hemithorax roughly parallel to the diaphragm and not

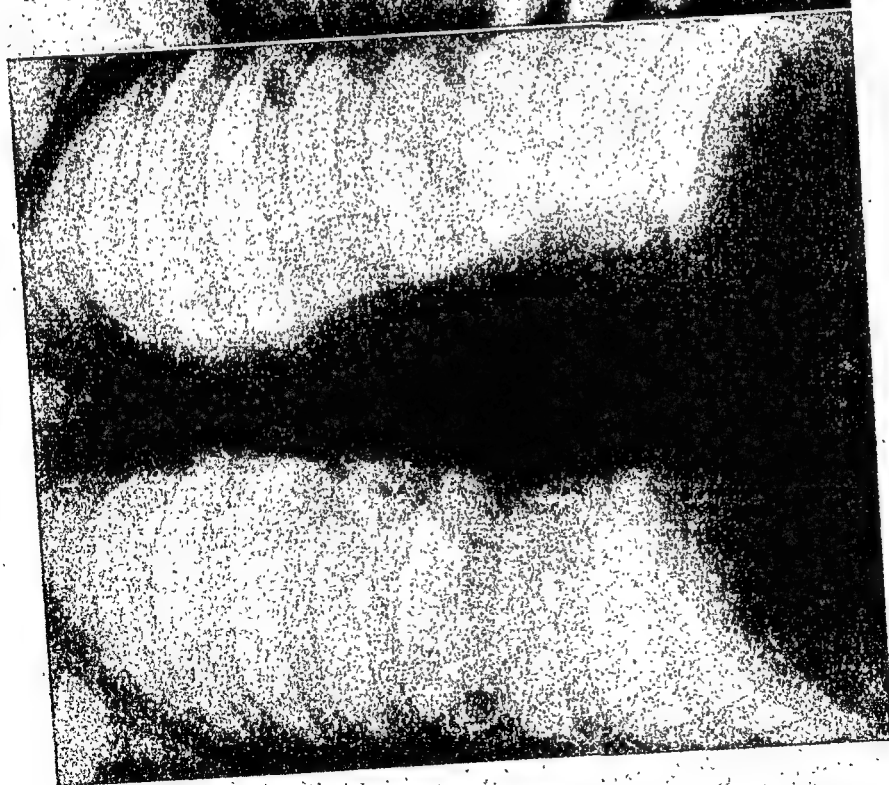


FIG. 142.—Atelectasis and consolidation of middle lobe.



Same case in lordotic position. The middle lobe is seen as a dense triangle with sharply defined borders.

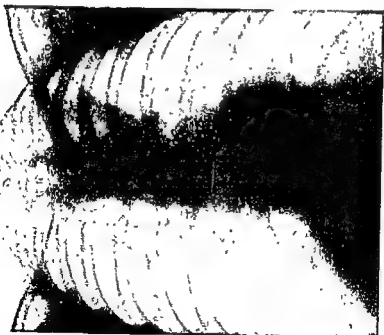


FIG 113.—Atelectasis of left upper lobe, following hemothorax. Chest previously normal. Spontaneous re-aeration, with normal radiographic appearance, occurred in two weeks.



FIG 144.—Lateral view. Segmental atelectasis in pectoral segment of right upper lobe. Child, 13 years. Post-pneumonic.

corresponding to any normal interlobar fissures. The writer has, on many occasions, noted these abnormal shadows. Recently in an asbestos worker with early signs of asbestosis, the shadow, not present five years previously, was found to have developed. In another case it was an accidental finding in a patient suffering from metastatic carcinoma in the spine, not bedridden, and showing no other sign of pleural or pulmonary metastasis. In another case it appeared after resolution of an atelectasis in a case of mediastinal neoplasm, but had not been present in a film taken prior to the collapse of the lung.

There is usually some associated elevation of the diaphragm and loss of aeration at the base. *Fleischner*, *Laurell*, and *Hulten* have drawn attention to these lines in patients suffering from, or convalescing from, acute abdominal conditions or fracture of the ribs. *Fleischner*, in a post-mortem of such a case, has found a transverse band of atelectasis running through the lung with a deep horizontal groove on the lateral surface of the lung corresponding to it. It would appear that these atelectatic lines follow prolonged elevation of the diaphragm, or long recumbence, or basal infection, but these are not essential factors.

Fleischner regards them as linear or disc-shaped areas of atelectasis, resulting from the prolonged occlusion of a small bronchus. The original pyramidal atelectatic lesion is pulled out into a flat form, being fixed medially by the bronchus, peripherally by the pleura, when the lung re-expands to its normal dimensions.

A possible, but as yet unproved, source of these lesions may be a local seam-like adhesion of an infolded portion of the visceral pleura, occurring during a period of incomplete expansion of the lung, as might occur in acute abdominal conditions. This might well give rise to a localised atelectasis, projecting into the deeper part of the lung from this region of the pleura. The true mechanism of production, however, is as yet unknown. In a few instances they may be accounted for by gross displacement of the interlobar fissure in which there is thickening of the interlobar pleura.

Occasionally an accessory interlobe separating the posterior accessory lobe, at the apex of the lower lobe, from the lower lobe is visible in the postero-anterior radiogram crossing the chest at mid-level. This can be distinguished from the horizontal lines just described by the lateral view. A normal main interlobe, displaced downwards and distorted by fibrosis, may also give rise to a similar shadow.

Haudek describes six cases with these horizontal or oblique shadows associated with disease of the liver, cholecystitis, peritonitis, etc. Autopsy in some cases showed pleural changes with deposits of fibrin. The diaphragm had been shown to be elevated slightly and its movement limited.

Udvardy has illustrated other cases of this condition secondary to abdominal conditions, and ascribes it to a direct transference of infection via lymphatics which permeate the diaphragm leading to subpleural pneumonic foci and pleural infection. The causative lesions were: (a) ovarian tumour, with ascites; (b) intestinal cancer; (c) cirrhosis of the liver with ascites; (d) appendicular disease; (e) pancreatitis; (f) renal infection.

CHAPTER XXI

EMPHYSEMA

THIS TERM indicates increase in air capacity of the lung or part of it (*Karsner*). It may be simple over-distension of the alveoli, but as a rule there is also rupture of the alveolar walls. It occurs in acute and chronic forms.

(1) Acute emphysema is observed in some cases of death from anaphylactic shock, by drowning or other forms of asphyxia; radiologically it may be observed in valvular obstructions by foreign body and in the form of acute vesicular emphysema.

(2) Chronic emphysema. Occurs in three forms: (a) Substantive—essential emphysema, (b) senile emphysema; (c) complementary emphysema.

CHRONIC ESSENTIAL EMPHYSEMA

Pathology.—This condition is also known as chronic, large lung, or "hyper-trophic" emphysema. The latter term is incorrect; there is no actual hypertrophy of the lung alveoli, but on the other hand an atrophic condition of their walls. It occurs in many forms of lung disease, especially asthma, bronchitis, tuberculosis, and silicosis. It frequently complicates, and may mask, bronchiectasis.

The cause is unknown, though repeated forcible coughing against resistance of tenacious secretions and reduction of the elasticity of the lungs with increasing age probably play some part. Toxic factors, e.g. alcoholism, syphilis, and inflammatory disease, may also predispose.

The lung is enlarged and does not readily collapse. The fusion of alveoli produces vesicles several millimetres in diameter, or large bullæ several centimetres in diameter. Bullæ are most commonly found at the anterior or inferior sharp margins of the lobes. They do not always communicate with bronchi. The elastic tissue of the lungs is reduced. Passive hyperæmia may be present in the later stage.

Radiology.—The radiological signs are: (1) Alteration in the shape and size of the chest and heart; (2) increased translucency; and (3) diminution in the change of translucency on deep breathing, and in the range of movement in the chest walls and diaphragm.

Kerley, in a thoughtful paper, points out that in the diagnosis of this condition radiology plays a leading rôle by bringing to light not only the emphysema itself, but also the underlying or associated pulmonary disease, to which the emphysema points, as a gastric incisura points to an ulcer. This paper describes the radiological findings in considerable detail.

The X-ray appearances in a typical case are as follows.

THE CHEST.—The transverse and antero-posterior diameters are increased. The ribs are more transverse and the diaphragms low. The apices are often dome-shaped. There is sometimes an associated kyphosis, and anterior bowing of the sternum in the lateral view; and there is an increase in the width and translucency of the space between the sternum and heart—the heart and aorta being displaced backwards (Fig. 145). The pectorals, scaleni, sterno-mastoids sometimes hypertrophy sufficiently to produce visible shadows.

THE HEART.—The heart shadow is usually small and narrow, due to a combination of three factors: (a) diminished venous return, so that the heart is actually smaller; (b) rotation of the heart to right, bringing the apex forwards; and (c) increased width of the thorax, with relative reduction of the heart width. The pulmonary conus may be prominent, owing to increased resistance to the pulmonary arterial circulation, and in later stages the right ventricle may be found enlarged. Owing to the descent of the diaphragm, air is visible beneath the heart in postero-anterior and lateral radiograms.



FIG. 145.—Emphysema. Lateral view showing increased antero-posterior diameter of chest, prominent sternum, and flattening of diaphragm.

A second type of emphysematous chest in which this narrow heart is not found occurs in obese individuals, usually middle-aged or elderly men. The chest is broad, the sternum high and prominent, and the spine often kyphotic. The high diaphragm pushes the heart up, so that it lies transversely, diaphragm

movements are diminished. As a rule the only underlying or associated pulmonary disease is a chronic bronchitis.

THE DIAPHRAGM.—This is usually one interspace lower than normal. The central tendinous part is horizontal. The cardio-phrenic angle becomes widened, approaching a right angle. The intercostal slips of origin of the diaphragm may be clearly visible.

THE LUNG MARKINGS.—The peripheral lung markings become less visible. Near the hilum the larger vessels stand out distinctly, partly because of an increase in their calibre, partly owing to emphysema of the alveoli surrounding them, which is sufficient to increase their contrast and definition, but incapable of causing over-exposure of such large structures, as it does in the case of the finer lung details. The lung markings of the upper lobes usually remain distinct, and may be increased if these vessels are carrying more than their normal contents of blood, or if there is an interstitial fibrosis.

Bullæ may develop in the course of chronic emphysema, or in any other type. They are described below.

SENILE EMPHYSEMA

This is also known as the "small lung" type. It occurs in elderly individuals, and is due to atrophy of the alveolar walls without absolute increase in the intra-alveolar pressure during expiration, normal respiratory pressure being sufficient to rupture the damaged alveolar walls.

RADIOLOGICALLY the appearances seen differ from the preceding only in degree. The lungs being small, the alterations in contour of the chest and in position of diaphragm and heart are less marked and may be unnoticeable. Respiratory variations in translucency and diaphragm movements are diminished because the chest is usually rigid and the lung inelastic. The finer lung markings are either normal or increased; the increase in the markings may in part be due to increase in the fibrous tissue of the lung stroma, but this interpretation in any given case is a matter of conjecture.

COMPENSATORY EMPHYSEMA

Compensatory emphysema is a passive dilatation of a whole lung, of one or more lobes, or of part of a lobe, and occurs when other parts of the lung are diminished in volume by fibrosis, atelectasis, or other disease. It is not compensatory in a functional, but only in a spatial sense, and is better termed "complementary" emphysema.

It may occur very rapidly and disappear as rapidly—for example in a case of collapse of one lobe from temporary blocking of its main bronchus—but may become permanent, in which case the alveolar walls lose their elasticity and become atrophic and a true permanent emphysema develops.

An extreme degree of compensatory emphysema is shown in Fig. 146; a museum specimen from a case of fibrosis of the entire left lung. This lung, invisible in the photograph, is represented by a small solid mass lying posteriorly. The heart, on the extreme left of the specimen, was in contact with the left chest wall. The right upper lobe filled both apical regions, and the right middle and lower lobes also appear in the left thorax.

RADIOLOGICALLY the striking feature is the apparent completeness with which the emphysematous lung may fill the chest. It is by no means uncommon to find, for example, a lung field of nearly normal size even in the presence of a completely collapsed lower lobe. The postero-anterior view in such a case may show the cupola of the diaphragm to be at a normal level, and the lateral costo-phrenic sinus well filled by the middle lobe. Careful inspection of the film will, however, show three things:

(1) The lung markings at the base do not pass downwards behind the diaphragm.

(2) In the lateral view the posterior costo-phrenic cul-de-sac is shallow and the posterior part of the diaphragm elevated.

(3) In both views, but more particularly in the postero-anterior, the visible lung detail, traced back to the hilum, is found to belong to upper and middle lobes, a fact which can be confirmed by the use of lipiodol.

Lipiodol will in such a case show that the lateral costo-phrenic sinus is occupied by a branch of the first ventral bronchus supplying the middle lobe. Similarly in atelectasis or fibrosis of an upper lobe the middle lobe vessels and bronchi arch steeply upwards towards the second or third interspace. Careful observation of such facts will prevent the overlooking of a compensatory emphysema, a mistake which is serious when, as often occurs, it is masking a bronchiectatic lower lobe. If the interlobar fissures are visible, they will usually be found to be convex towards the shrunk portion of the lung and away from the emphysematous lobe.

If the lung is greatly enlarged by compensatory emphysema, it often dis-

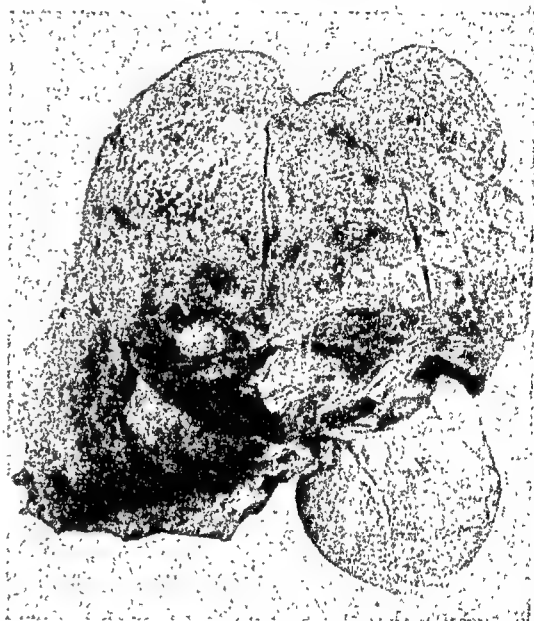


FIG. 146.—Compensatory emphysema. Right lung filled the entire chest, and both apices were formed by right upper lobe, which had become moulded into both apical domes. Right middle lobe reached across chest to left chest wall, above the heart. The fibrosed left lung, lying posteriorly, is invisible in the photograph. (Specimen, Path. Mus. Manchester University.)

places the pleura across the midline, and its edge appears on the opposite side of the sternum. Usually the ballooning is anterior to the heart, as the lateral view clearly demonstrates. If the area of lung affected by compensatory emphysema is large, the changes in ribs previously described will occur. In-



FIG 147—F 48 Surgical emphysema extending from mediastinum into neck as a result of perforation of œsophagus by swallowed meat bone.

creased translucency of the lung and a fanning out of the lung markings, with obliteration of the finer lung details, are also seen.

Pulmonary collapse (neoplastic or otherwise), tuberculosis, fibrosis, and bronchiectasis are the conditions in which compensatory emphysema most commonly occurs. It is stated that it does not accompany the "epituberculous" lesions of childhood (Kerley).

ACUTE VESICULAR EMPHYSEMA

This is gross localised alveolar dilatation, which may give rise to clearly defined ring shadows. These occur with some frequency in the neighbourhood of the consolidated area in children with pneumonia, and simulate cavities. They may disappear rapidly or persist for months.

BULLOUS EMPHYSEMA

Bullæ may occur in any type of emphysema. They are shown as ring-shaped areas if single, or as polyhedral translucent areas separated by fine linear walls where several bullæ lie in contact. They are sometimes subpleural, sometimes interstitial. Lipiodol cannot be made to enter them, a point of value in their differentiation from congenital lung cysts—which can usually be filled. They may simulate tuberculous cavities. Bullæ may be observed on the surface of the lung in pneumothorax, especially if the lung is carefully screened at varying angles. In a series of fifty-five cases of spontaneous non-tuberculous pneumothorax, *Kerley* was able to demonstrate them in eight cases. It is probable that the large pleural rings sometimes found in pneumothorax are actually emphysematous bullæ. It was shown by *Laurell*, by experiments in calves, that emphysematous blebs and bullæ cause ring-shaped shadows in the radiogram, the marginal density being due to a surrounding zone of atelectatic alveoli.

INTERSTITIAL AND SUBCUTANEOUS EMPHYSEMA

Air may enter the *interstitial* tissues of the lungs if the alveoli are torn by needle punctures, rib fractures, penetrating wounds, or other injury, or after tracheotomy or perforation of the bronchi. Violent coughing in children may have the same effect. The air is present in bubbles in the interstitial tissue and underneath the pleura between the anatomical lobules. It may extend into the mediastinal tissues and may follow the cervical fascia into the neck, and may even extend over the chest and abdomen as subcutaneous emphysema.

Subcutaneous emphysema is more commonly seen as a result of admission of air to the pleural cavity by needle puncture, with or without actual penetration of the lung, with subsequent expression of the air during coughing or breathing.

RADIOLOGICALLY subcutaneous emphysema is frequently seen in the form of numerous air-containing translucent patches in the subcutaneous tissue. On palpation, the feeling of crepitation is as characteristic as the radiological picture. Occasionally in war-time the writer has seen a similar picture as a result of gas infection without pulmonary involvement.

Interstitial and mediastinal emphysema are very rarely seen radiologically. *Assmann* describes a case in which mediastinal emphysema occurred as a result of intense dyspnoea in a case of bronchiolitis obliterans. The radiogram showed along the borders of the mediastinum a broad translucent stripe contrasted with the opaque lung field, of finger breadth, which was confirmed by autopsy. This revealed emphysema consisting of air vesicles in the loose tissue beneath the mediastinal pleura, to which it had spread from the roots of the lungs.

Lipiodol in Emphysema.—Lipiodol filling shows the bronchi to be fanned out and separated widely from one another. The smaller bronchioles often fail to fill, or may do so irregularly. A beaded appearance is due to slight localised

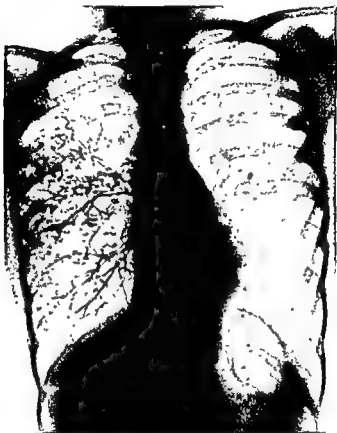


FIG 148 —Bilateral lower lobe collapse, with marked compensatory emphysema of the remaining lobes. Lower lobes contracted to small triangles behind the heart shadow. On the right side the upper and middle lobe bronchi are outlined with lipiodol.

dilatation of the small bronchi at the points where they are unsupported by cartilage.

Christopherson finds that the alveoli fill more readily if emphysema is present. In the absence of emphysema the filling of the terminal bronchioles and alveoli is stated by this author to be dependent upon the preponderance of sympathetic (dilator) or vagal (constrictor) nervous control. Minor degrees of dilatation of the medium-sized bronchi are demonstrable in asthma.

CHAPTER XXII

PNEUMONIA AND FIBROSIS

ACUTE LOBAR pneumonia is characterised by consolidation of one or more lobes of the lung. It is caused by pneumococci which enter the lungs through the air passages. *Blake and Cecil* (1920) found experimentally that pneumococci pass through the bronchial walls in the hilum into the interstitial tissue, where they spread outwards along the perivascular and peribronchial lymphatics, this cellulitis being followed by invasion of the alveoli from without. The hilar regions are the first to consolidate. *Schöbl and Sellards* (1926) also found this "wave" of progress outward. *Gaskell* (1925-7) made experimental intrabronchial injections into rabbits, passing a catheter into a bronchus and introducing pneumococcal cultures. By varying the virulence of the cultures used, various degrees of pneumonia were obtained, the less virulent giving bronchopneumonia; the more virulent, lobar pneumonia; and the highly virulent, septicæmia.

Types of Pneumococcus Pneumonia.—By serological methods, three main types of pneumococcus (I, II, and III) are distinguished. A fourth group (IV) is a category in which are placed pneumococci which are not identifiable with Types I, II and III. Pneumonia due to Type IV is usually mild, and these are the pneumococci found in healthy carriers or patients with chronic catarrh of the upper air passages.

Types I, II, and III are more virulent but less widely distributed. "Type I" pneumonia is the classical variety with sudden onset and well-defined physical signs. These cases show early consolidation, and spread from hilum to periphery; X-ray findings show rather more widespread lesions than can be detected clinically, and in over 50 per cent. more than one lobe is affected. Consolidation is maximal on the seventh to eighth day.

Origin and Spread of Lobar Pneumonia.—In children the disease often appears to commence peripherally and to spread to the rest of a lobe, until ultimately the shadow reaches the hilum. It would appear that the pneumococci penetrate farther along the bronchi before piercing the bronchial wall in such cases.

The classical stages of congestion—red and grey hepatisation—cannot be clearly distinguished in the radiograms. The congestive stage shows only an increase of the vascular shadows. It lasts but a few hours. Red and grey hepatisation show no radiological difference from one another. The shadow of pneumonia, once fully developed, endures until the crisis: shortly after the crisis there is visible evidence of resolution in the X-ray.

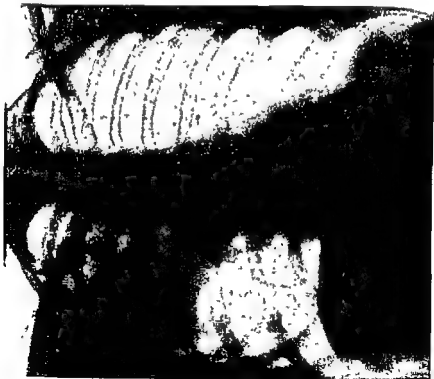


FIG 149.—Lobar consolidation (pneumonia) of right upper lobe, limited below by the horizontal fissure.



Lateral view. The pneumonic lobe is limited below by the main fissure behind, and the horizontal fissure in front.



Lateral view to show limitation of the consolidation by the interlobar fissure: lower lobe normal.

FIG. 150.—Pneumonic consolidation of the left upper lobe: its apical segment is not affected.

LOBAR CONSOLIDATIONS : RADIOLOGICAL APPEARANCES

Sante, in 1923, gave an exhaustive account of the radiological appearances of lobar pneumonia in a series of 152 cases, examined radiologically at intervals of one to three days. He pointed out the dependence of the radiological findings on the arrangement of the lobes and fissures.

In Right Upper Lobe Consolidation, the whole upper lung field is opaque. The lower border in the postero-anterior radiogram is well defined, owing to limitation at the horizontal interlobe. If an element of collapse is present, the interlobe is convex upwards, but if the consolidated lobe is massive, without collapse, the horizontal interlobe may be bulged downwards. In the lateral view the shadow is shaped as in Fig. 149, and it will be noted that the well-defined bounding line between the posterior part of the upper lobe and the upper part of the lower lobe is only to be seen in this view, since in the postero-anterior view it is oblique to the rays.

In Left Upper Lobe Consolidation, the upper and inner three-quarters of the lung field is opaque, and the lower and outer border of the opacity follows the line of the incisura of the oblique fissure. This border is not sharply defined in the postero-anterior view because this margin of the lung is thin and triangular in section, and casts a shadow of gradually diminishing density towards its lower edge. The costo-phrenic angle is normally translucent, this part being occupied by the lower lobe. In the lateral view the appearances are very characteristic—a sharply defined oblique line of the main fissure divides the antero-superior opaque upper lobe from the postero-inferior lower lobe.



FIG. 151.—Consolidation of right middle lobe, lateral view. F. 12. Chronic pneumonia, possibly tuberculosis. Resolved six months later.

In Middle Lobe Consolidation, the upper border is bounded by a horizontal line, usually convex upwards. The shadow is triangular; its lower and outer margin follows the line of the incisura of the main fissure on the surface of the lung, and, as in the case of the left upper lobe consolidation, fades out as this line is approached, since this margin of the lower lobe is also triangular in section. The costo-phrenic angle occupied by the lower lobe is clear.

In Lower Lobe Consolidation.—In the postero-anterior view the lower two-thirds of the lung field is opaque. The opacity is homogeneously dense

at the base and fills the costo-phrenic angle. In the upper third of the lung field the margin becomes ill defined, since the upper margin of the lower lobe tapers off in this direction. In the lateral view the oblique fissure divides the lung field into an antero-superior translucent half and postero-inferior dense half. If atelectasis is combined with the consolidation, the oblique fissure is displaced backwards and downwards, and the posterior part of the diaphragm raised on the affected side.

These observations of *Sante* have been endorsed by all observers, and are well known. More difficult to evaluate and to diagnose radiologically are partial pneumonias regarding which the writer offers the following observations.

PARTIAL PNEUMONIAS

The pneumonic process may affect only one portion of a lobe at the time of examination and subsequently spread to the remainder of the lobe, or it may be limited from the beginning and may remain limited throughout the disease. Such partial pneumonias, of whatever aetiology, show pyramidal areas of shadowing, and two projections may be necessary in order to show their pyramidal shape and true nature. They usually occupy the distribution of a definite branch or branches

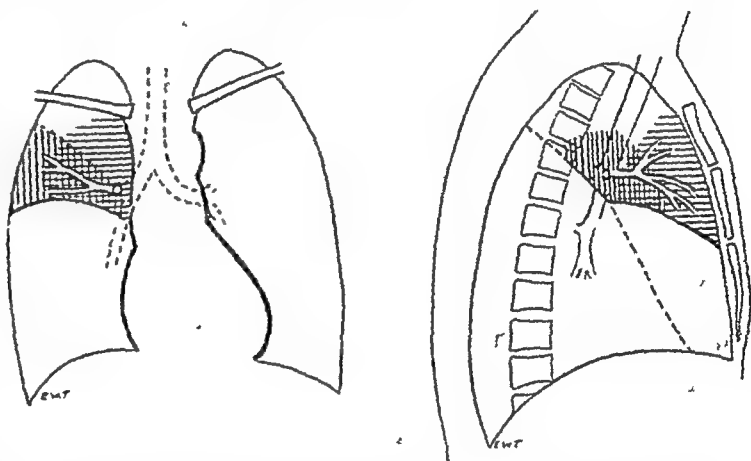


FIG. 152.—Diagram: Consolidation in the pectoral and axillary segments of the right upper lobe in anterior and lateral projections.

of the main bronchus supplying that lobe and commonly lie near the interlobar margin. If atelectasis is present as well, the interlobar fissure is then convex towards the opacity. On the right side in the postero-anterior view consolidation in the distribution of the *pectoral branches* of the *right upper lobe* bronchus is shown as a triangular shadow, with its base continuous with the right border of the mediastinum, its apex outwards, its lower border sharply limited to the horizontal interlobe. Consolidation limited to the *posterior apical branch* produces a somewhat ill-defined shadow, which may or may not show a clear-cut lower margin in the postero-anterior view. Usually this margin is not clear-cut, since the adjoining fissure is oblique. Consolidation limited to the distribution of the *axillary branches* of the *right upper lobe* is triangular with base at the axillary wall, apex towards

the hilum, and is limited below by the horizontal interlobe. The appearances given by these partial consolidations in the postero-anterior and lateral view are shown diagrammatically in Fig. 152. Partial consolidations limited to

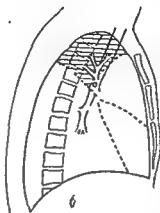
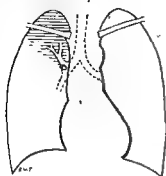


FIG 153.—Diagram : Consolidation in the apical segment of the right upper lobe in anterior and lateral projections.

the *ascending apical branches* of the upper lobe show a triangular opacity, with base directed towards the supraclavicular region, apex at the hilum, and inner border continuous with the mediastinal shadow (Fig. 153). These have to be distinguished from complete atelectasis or fibrosis of the upper lobe and from consolidation in an azygos lobe. (a) From atelectasis or fibrosis of upper lobe : In this condition there is : (1) a "fanning out" of the bronchial shadows of the middle and lower lobes on the right side or lower lobe on the left side ; and (2) compensatory emphysema of those lobes. (b) From consolidated azygos lobe : the latter azygos lobe usually has a convex outer border.

Partial consolidations limited to the *first dorsal branch of the lower lobe bronchus* shows in the postero-anterior view a somewhat diffuse circular shadow, not sharply defined on either side, which lies to the outer side of the upper hilar region. In the lateral view it is sharply defined above by the upper part of the oblique fissure, less well defined below, and triangular in shape ; with its base at the posterior wall, apex at the hilum (Fig. 154). If this region exists as a separate

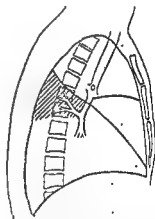
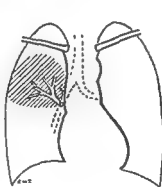


FIG 154.—Diagram : Consolidation in the apical segment of the right lower lobe in anterior and lateral projections.

exists as a separate



Lateral view. The consolidated segment is bounded anteriorly by the lower part of the main fissure.

FIG. 155.—Pneumonic consolidation in anterior basal segment of right lower lobe.

lobe, the triangle is sharply defined below by the accessory fissure. Partial consolidations in the *remaining area of the lower lobe* show ill-defined shadows in the postero-anterior view. In the lateral view they have their apices directed towards the hilum, and their bases downwards and directed either anteriorly, vertically, or posteriorly according to the bronchus involved. When the *whole distribution of the lower lobe bronchus* is affected with the exception of the first dorsal branch, a characteristic broad triangle is seen in the lateral view.

Differential Diagnosis.—If reliance is placed upon a plain postero-anterior film, it may be quite impossible to differentiate these partial consolidations from interlobar effusions, since these effusions lie in the fissures bounding the lobes. An effusion in the upper part of the main interlobar fissure may show in the postero-anterior radiogram sharp limitation below at the junction of the fissures, and present a shadow very similar to a consolidation of the lower part of the upper lobe. An effusion in the lower part of the right oblique fissure is difficult to differentiate from middle lobe consolidation. The lateral view nearly always clears up the diagnosis. A posterior parietal effusion on the back of the chest wall, in the postero-anterior view, shows a diffuse shadow often mistaken for a lower lobe consolidation. In the lateral view its convex anterior border is characteristic. Interlobar effusions are relatively rare, consolidations very common.

COURSE OF LOBAR PNEUMONIA

Davies, Hodgson, and Whitby, in an important review of 110 cases of pneumococcal pneumonia intensively studied during the whole course of their disease, found few early cases in the group investigated. Nine cases were examined radiographically in the first forty-eight hours and sixteen in the first seventy-two hours. The first change noted was a perihilar density which rapidly reached the periphery, often within twenty-four hours. There was usually evidence of some diminution of the lung volume, namely elevation of diaphragm to the extent of one or two rib spaces, narrowing of the intercostal spaces, and occasionally displacement of the heart and mediastinum towards the affected side. In a few cases the periphery was first affected and the consolidation spread towards the hilum. Physical signs in these cases were usually absent until the periphery was involved.

The maximum opacity was reached in four to seven days. The diminution of volume of the lung noted by these workers paralleled the experimental work of *Terrell, Robertson, and Coggleshall*, and is extremely interesting. The association of some degree of atelectasis with pneumonic consolidations is a matter of fairly common observation. It has been stated that this can only occur in the resolving stage, since the alveoli when filled with exudate cannot collapse, whereas absorption of exudate before the bronchial channels are reopened would leave the alveolar walls more closely approximated, and so

l to diminution in the lung volume. The clear evidence of *early* diminution of lung volume in the series studied by *Davies, Hodgson, and Whitby* shows that an element of atelectasis may certainly be present from the first. This might follow plugging of the bronchi by plugs of mucus (*Coryllos* and *Birnbaum*), though the evidence that this occurs in lobar pneumonias is not altogether convincing. Obstruction of large bronchi by bronchial plugs usually produces a massive collapse of a lobe or part of a lobe, with radiological appearances which are unmistakable. These are rarely seen in lobar pneumonias, in which the diminution of volume is usually moderate as a rule. It would appear that the atelectasis is more uniform in nature, and this points to a more peripheral block, immediately preceding or coincident with the outpouring of exudate into the alveoli. Such a block might be produced by exudate in the respiratory bronchioles, the walls of which are studded with alveoli, or in the finer ducts supplying groups of terminal alveoli. Rapid absorption of the small quantities of air still present in the alveoli at this stage would lead to a definite uniform atelectasis, radiologically indistinguishable from the true consolidation (due to intra-alveolar exudate) with which it is mingled.

In the series just mentioned the following points were noted in the various types of pneumonia.

Type I Pneumonia.—An interesting finding in the series referred to is the enlargement of hilar glands which is visible as soon as resolution begins, and not before (*Graham Hodgson*): and this enlargement indicates that resolution has begun. The glands are globular in outline. Resolution is complete in periods varying from ten to fifty days, with an average of twenty days, and is more rapid in the cases showing glandular enlargement. The denser consolida-

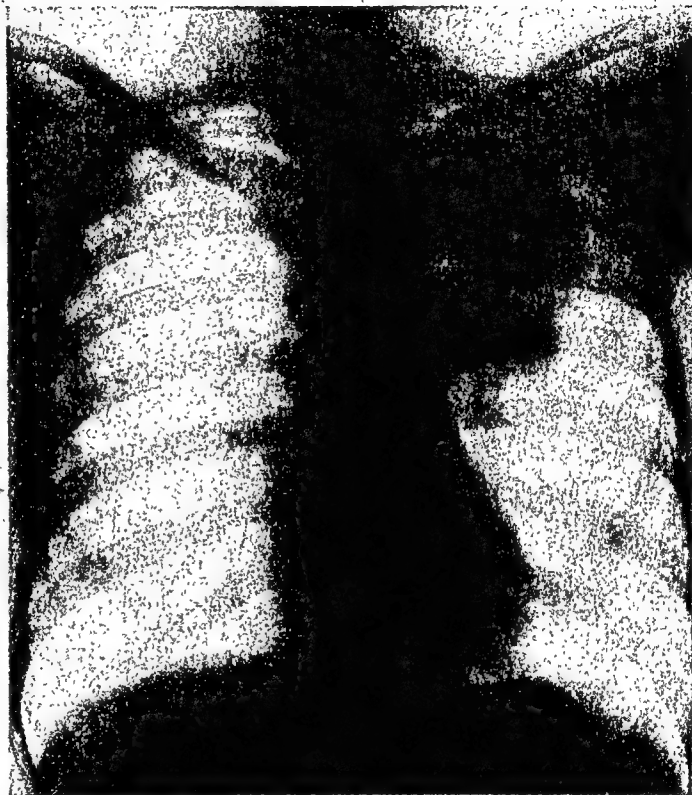


FIG. 156.—Acute pneumonia, left upper lobe.

tions are usually associated with severer symptoms and course. Serum treatment shortens the febrile period and induces an early crisis, but does not materially hasten resolution.

Type II Pneumonia.—This is more severe than Type I, with marked toxæmia, sudden onset, and well-defined signs of consolidation. The only difference in radiological appearances is a tendency in Type II to a permanent slight increase in the density of the pulmonary stræ.

Type III Pneumonia.—This type attacks elderly and debilitated people and has a high mortality. The onset is less sudden and the course less well defined. The opacity is patchy rather than homogeneous, and usually peripheral. The diaphragm is not elevated, as a rule, and the course is prolonged. Resolution varies from thirteen to seventy-two days.

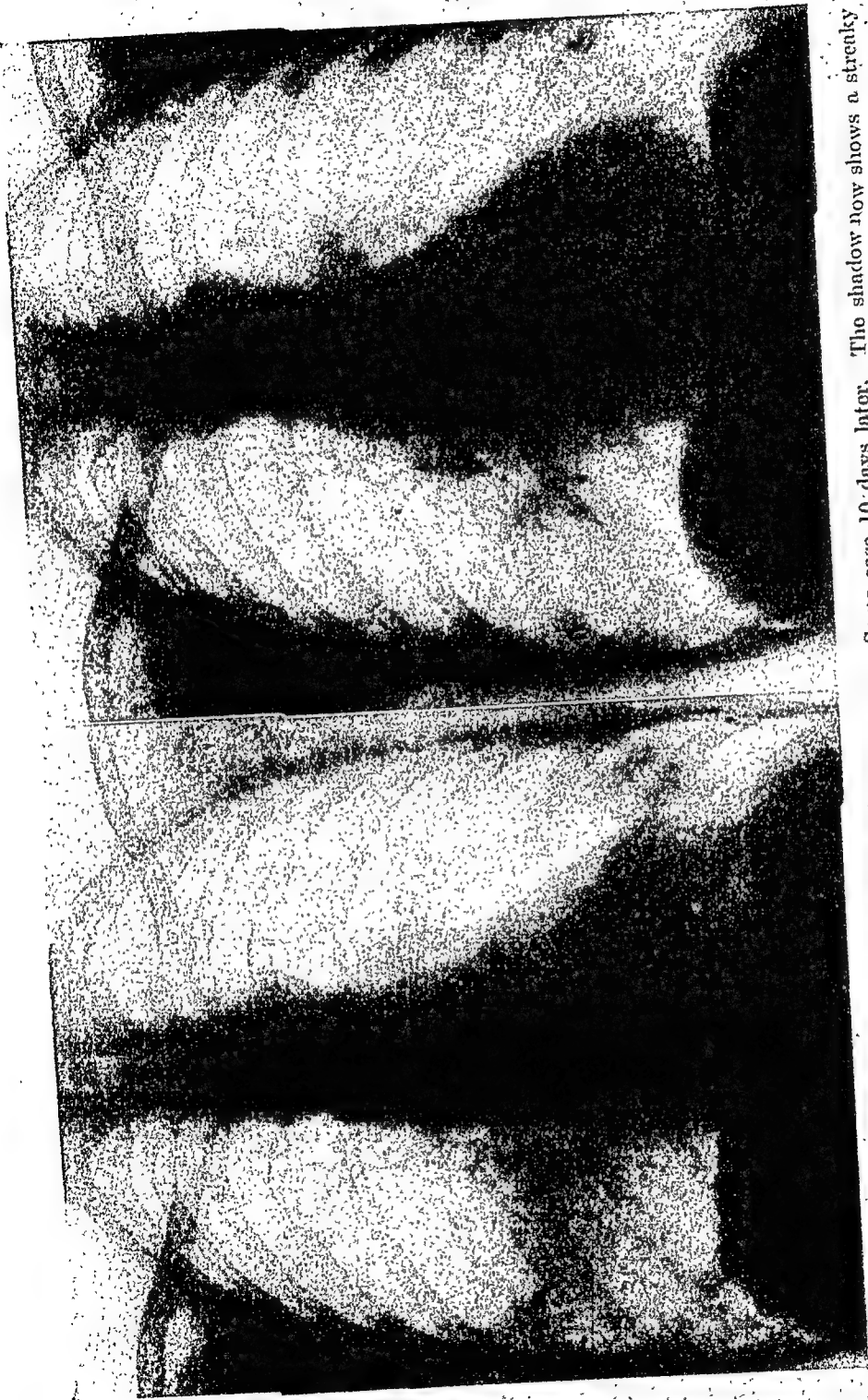
Group IV Pneumonia.—Cases in this group are well defined clinically and the radiological appearances also vary. The opacity is moderately dense, and in one reported case (*Graham Hodgson*, loc. cit.) resembled miliary tuberculosis. In two cases an appearance suggesting cavitation was seen, and disappeared during the course of the illness. *Terrell, Robertson, and Coggleshall* found similar cavities in the resolving lungs of dogs, and showed that they were due to dilated atria or air sacs. Some writers consider them to be areas of bullous emphysema; others believe that they are true cavities ("aputrid necrosis"—*Rabin*).

Resolution.—The opacity clears up first at the periphery, and the clearing extends inwards until the hilum region is reached. Empyema occurring during the stage of full consolidation cannot be distinguished radiologically, but may be inferred if the heart is becoming displaced to the opposite side.

Central Pneumonia.—Many so-called hilar or central pneumonias can be shown by lateral views to be due to a partial involvement of the lower lobe which is in the postero-anterior view superimposed upon the hilar region. The writer agrees with *Ude*, that central pneumonia "is in many cases a misnomer."

Resolving Pneumonia.—Resolving pneumonia shows a characteristic appearance which is easily recognised. The opacity gradually diminishes, but the affected area is seen to be permeated by striations and fine lines of increased density for a time, an appearance which has been ascribed by various writers, (a) to thickening of the lymphatics, which become clogged with absorbed exudate and inflammatory cells; (b) to localised strands of peribronchial and perivascular atelectasis. As a rule these appearances clear up completely. In an uncomplicated case the chest may appear normal a week after the crisis. (See Fig. 157, opposite.)

During resolution the interlobar margins tend to clear up more slowly than the remainder of the affected lobe, and the consolidation tends to linger in the region of the interlobar fissure. This is often brought out very clearly in lateral and oblique views, and a residual line of interlobar sclerosis is often visible for months after the patient is clinically well.



Same case 10 days later. The shadow now shows a streaky appearance. Alveoli becoming re-aerated but lung stroma in the affected region shows dense lines due to congested lymphatics.

Fig. 157.—Resolving pneumonia, right lower lobe.

Delayed Resolution.—It is clear from the figures quoted above from *Davies*, *Hodgson*, and *Whitby's* observations that the time taken for resolution is very variable. In delayed resolution the homogeneous shadow may continue unchanged for a considerable time, but as a rule it gradually loses its homogeneous appearance and shows a cloudy mottling, due to patchy re-aeration of the lung. Gradually the shadow becomes less dense and is permeated by a network of strands. In some cases delayed resolution may be observed to pass gradually into fibrosis: the affected part shrinks and bronchiectatic cavities may develop within it.

Diaphragm.—The diaphragm is often raised, and is immobile during the acute stage. In lower lobe pneumonias it may continue to be elevated for several weeks after resolution.

SEQUELÆ OF PNEUMONIA

Empyema.—Cases of acute pneumonia are not as a rule subjected to X-ray examination, for obvious reasons. The principal use of radiology is the investigation of delayed resolution and commencing empyema and other post-pneumonic complications. The occurrence of empyema is shown by a typical shadow of pleural effusion, in children often lamellar in type, or in the form of a band extending up the whole length of the parietal wall. In other cases, a typical pleural curve is present.

Very careful search must be made in all planes for indications of localised encysted pleural effusion, particularly common on the lateral wall and in the posterior cul-de-sac, less common in the interlobar fissures. With generalised effusion, the heart is nearly always displaced towards the opposite side. In children the costo-phrenic angle may appear somewhat translucent, even in the presence of considerable effusion. This was noted in 28 per cent. of 325 cases of pleural effusion in children studied by *Carty* and *Liebman*.

No great difficulty attaches to the diagnosis of empyema as a rule, but where there are patches of residual consolidation it is by no means easy to say how much of the shadow is due to fluid and how much to consolidation. In cases where the whole or greater part of one base is opaque, an effusion may be present but undiagnosable. Stress must be laid upon the displacement of the heart and bulging of the mediastinum towards the other side, which never results from pneumonic consolidation, on widening of the rib spaces (fluid), and on the position of the diaphragm, which is displaced downwards by fluid but unchanged or displaced upwards by a pure pneumonic process. Perhaps the most important point is that, in empyema, especially in children, rapid changes occur from day to day, and a second examination will usually clear up the diagnosis.

Thickened Pleura.—Fibrinous exudate, in a thick layer, often covers the parietal pleura. After drainage of an empyema, this layer is seen as a band of shadow in the radiogram on the inner chest wall, often reaching to the apex, its

inner edge well defined by the surgical pneumothorax : it may be as much as an inch in thickness. The corresponding exudate may be visible upon the collapsed lung. It is obvious that it is difficult, prior to operation, to say how much of the parietal shadow is due to fluid and how much to fibrinous exudate. In some cases there is no appreciable fluid, the entire shadow being due to fibrin. In this case the heart is not displaced, or displaced to the same side. In case of doubt, the effect of change of position upon the shadow must be tested. Pleural effusions change their position, usually promptly, but sufficient time should be allowed for this to occur. The lateral lying position is useful and produces a more obvious alteration of contour (*Lenk*).

PNEUMONIA IN CHILDHOOD

Pneumonia in children has been described by *St. Engel* and *Samson*. It may be acute lobar, or bronchopneumonic. In general, pneumonias in childhood differ from the adult type as follows :

(1) The shadow is, as a rule, less massive and very frequently partial, remaining confined to one part of a lobe. The pneumonic area may be small from beginning to end of the disease. (2) There is a marked tendency for collapse and pneumonia to be combined, especially in younger children. The younger the child, the greater the tendency to collapse. (3) The paravertebral segments are often involved alone, especially in infants. On the right side the upper lobe is more frequently affected ; on the left, the lower. Radiologically the shadow is often confined to the hilar region, with which it fuses. If the right upper lobe is affected, the usual abrupt limitation by the horizontal middle fissure is often absent. (4) There is a marked tendency to pleural complications—either lamellar pleurisy, overlying the lesion, or frank empyema.

In infancy the commonest type is paravertebral pneumonia. In early childhood the commonest type is lobular bronchopneumonia. In later childhood and school age the commonest type is frank lobar pneumonia.

Course of Lobar Pneumonia in Childhood.—After a more or less definite prodromal stage, there is an abrupt onset, with high temperature, which lasts usually five to seven days, and then rapidly becomes normal after the crisis.

The pneumonic shadow is first seen, as a rule, about the first to fourth day. Early involvement of the hilar glands is common in children, and the shadow often extends outwards from enlarged tracheo-bronchial and paratracheal glands. In the lower lobes a rounded paravertebral shadow is often seen in the first stage, commencing in the apex of the lower lobe, which in the course of a few days spreads to the lower lung field. It is evident that the radiological differentiation from a primary tuberculous infection is difficult. In both conditions one may find a homogeneous shadow in the lung with enlarged hilar glands. The essential difference lies in the rate of development and resolution. In pneumonia, the course is measured in days ; in primary tuberculosis, in weeks or months.

Origin in the hilar region is the rule in lower lobe pneumonias; but in upper lobe pneumonias, the exception. In upper lobe pneumonias the extreme apex often escapes. The shape of the shadow in total lobar involvement is the same as that seen in adults. The cases must be studied in lateral as well as in the postero-anterior views. Pneumonia may occur in an azygos or in an infracardiac lobe and be confined to it. Occasionally in right upper lobe lesions the azygos lobe escapes and is shown as a clear apical area with convex sharply limited outer border.

BRONCHOPNEUMONIA

Pathology.—The extension of an inflammatory process from a bronchitis of the smaller bronchioles to the units of lung parenchyma which they supply constitutes bronchopneumonia. The disease thus tends to be lobular in character. The term in practice covers all non-specific pneumonias.



FIG. 158 —Section infant's lung $\times 3\frac{1}{2}$ Lobar type of bronchopneumonia. Note the interlobar septa demarcating consolidated lobules from one another and from the other lobules.

The causative organisms vary widely and include streptococci, staphylococci, pneumococci, Friedländer's bacillus or other mouth organisms, bacillus coli, b. pestis, and others. The disease occurs most frequently in infancy and old age. It complicates the acute exanthemata, especially measles: it results from inhalation of irritating gases—chlorine, ammonia, bromine, and war gases—or aspiration of septic material. Tuberculous bronchopneumonia is not uncommon as a result of bronchogenic spread from an existing lesion. Lobar pneumonia may also occur as part of a septicæmia, in which case the disease is blood-borne and the bronchioles are not primarily involved.

The anatomical lesion consists of patchy areas of consolidation, usually bilateral and in the lower lobes. The unit involved may be an anatomical lobule, but as a rule the areas are ill-defined and without definite lobular boundaries and vary much

in size. Pus, mucopus, or fibrin may be present in the affected alveoli and the bronchioles are inflamed. In some forms, especially the hæmolytic streptococcal forms, there is a pronounced tendency to hæmorrhage.

Resolution occurs as in ordinary pneumonia, but there may be organisation of the exudate and fibrosis, or abscess or gangrene may occur as a complication.

Onset is usually insidious and the course and duration very variable. Most cases resolve by lysis. In an uncomplicated case resolution may occur in a few days.

Radiological Appearances.—Simple lobular pneumonias show mottled shadows, usually basal. They usually appear first in the paravertebral regions, and later tend to be more numerous and closely packed in the region bordering the heart. They are diffuse, with ill-defined margins, owing to surrounding hyperæmia. They vary in size from a quarter to an inch. Very often they are confluent and may fuse together till they occupy an entire lobe (pseudolobar form).

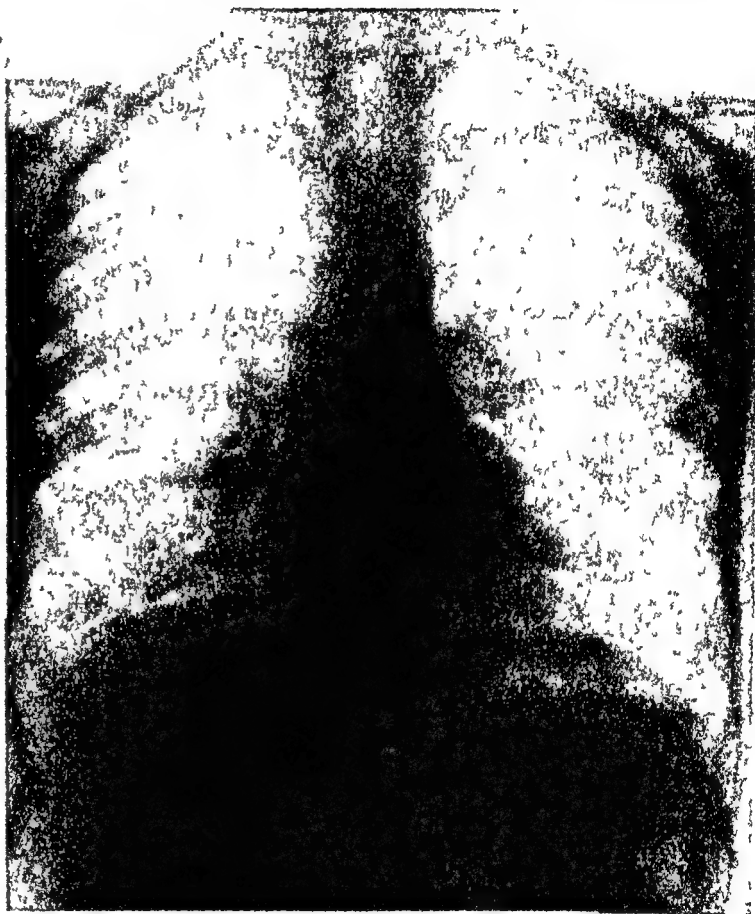


FIG. 159 —Acute bronchopneumonia.

Lobular pneumonia may occur in the upper lobes and closely simulate tuberculosis. The writer has seen several such cases in which a diagnosis was only rendered possible by the relatively rapid change in the appearances, which cleared up in a few weeks. It is a fairly common type in children. A widespread, symmetrical distribution of relatively well-defined lobular infiltrations (*miliary bronchopneumonia*) of nearly even size may even simulate a pneumoconiosis, as far as pure X-ray appearances are concerned.

From miliary tuberculosis the condition is differentiated by the larger size and less regular shape of the foci, and, as a rule, by the non-involvement of the apices. Miliary bronchopneumonia is especially common in measles.

Suppurative bronchopneumonia tends to become confluent and to lead to gross abscess formation, cavitation, and pleural complications, especially empyema. Bronchiectasis may also ensue. This type may, however, resolve completely.

The radiological appearances of cavitation are unmistakable when air has entered the cavity. Until this occurs the diagnosis cannot be made.

In all forms of bronchopneumonia the hilar shadows are increased owing to tumefaction of the hilar glands, hilar periadenitis, and hyperæmia.

INFLUENZAL PNEUMONIA

Epidemic Influenza.—The influenza epidemics of 1918–20 showed many types of lung involvement. Six modes of invasion have been described (*Sante*)

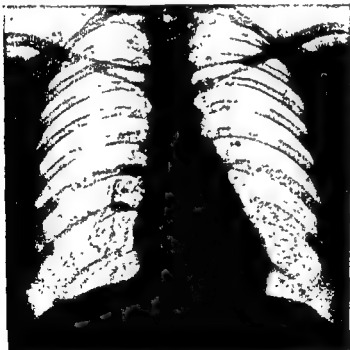


FIG. 160.—Influenzal bronchopneumonia

in which the radiological picture has been correlated with the clinical and autopsy findings.

(1) By numerous small infiltrations which coalesce to form small discrete areas of bronchopneumonic consolidation occupying all lobes, but more marked in the lower a classical bronchopneumonia commencing as a bronchitis and due to the specific influenza organisms.

(2) Similar infiltrations, at first small later coalescing to form a consolidation of an entire lobe (pseudolobar form)

Different lobes may be invaded one after another. The pseudolobar type is the commonest, and resolution often occurs without further spread.

(3) Blotchy infiltrations, coalescing to form a general haziness over a limited part or parts of the lung field, not confined to one lobe. Autopsy shows

a diffuse pneumonitis resembling that of a streptococcal septicæmic pneumonia affecting all components of the lung. Spread is rapid and the prognosis serious. Death may occur in a week. This form is rather uncommon and due to a mixed infection with streptococci.

(4) A hilar type, commencing at the hilum and rapidly sweeping to the periphery, usually running a rapid course, and attended with high mortality. Abortive forms, however, occur in which the opacity after reaching a certain stage resolves, and the disease terminates usually by crisis. These favourable forms are probably pneumococcal. Autopsy in the more severe types shows a zone of purulent and hæmorrhagic infiltration surrounding the larger bronchi and rapidly extending towards the periphery. Usually streptococcal, this type has a high mortality.

(5) A basal form, in which the opacity appears first in the most dependent parts of the lung and very rapidly progresses upwards by continuity. A fatal type is usually associated with streptococcus or streptococcus hæmolyticus. The first infiltrations in the costo-phrenic sinus may spread to involve the lower half of the lung within twenty-four hours and death may



FIG. 161.—Septic bronchopneumonia.

occur within forty-eight hours from the onset; clinically excessive prostration, high temperature, and delirium are present from the outset. Fluid is not present in the pleural cavity.

(6) True lobar pneumococcal pneumonia is occasionally observed.

Sante describes the pneumonias of this epidemic in general as the "most lawless of the chest infections." Radiologically spread or extension can be noted from day to day. Abscess formation, frequently of the small multiple type, is common. Resolution usually requires from six to eight weeks.

Misske and *Sylla* studied 126 cases during the influenzal epidemic of 1928-9

—including eighty catarrhal and thirty-three pneumonic. The Pfeiffer bacillus was present in 65 per cent. of these cases. Their radiograms showed a great variety of appearances, difficult to classify, including catarrh, bronchopneumonia (miliary, lobular, and pseudolobar), bronchiectasis, fibrosis, empyema. They rightly emphasise the resemblance which an incompletely resolved influenzal pneumonia may bear to a bronchial carcinoma.

Apart from epidemics, atypical pneumonias are seen in the winter months. The following forms have been described :

(1) A mild form, with moderate pyrexia, lasting a week or ten days. Radiologically bronchopneumonic foci are found in one base, rather larger than in ordinary bronchopneumonia. They do not coalesce and clear up rapidly. They are probably due to a mild specific influenzal infection.

(2) A similar mild form in which the loss of translucency is confined to one lobe. The opacity is not complete and does not totally obscure the lung markings. It clears up in a week to a fortnight.

(3) A severe form with acute onset, with physical signs of bronchitis, but severe constitutional symptoms. The typhoid state may supervene. Radiologically, in the early stage, there is diminished translucency and congestion of both lungs, without distinct patches of bronchopneumonia. Pyrexia may persist for months, and during convalescence the opacity disappears, but is replaced by a network of fine lines spread throughout the lungs, including the apices, ascribed by *Assmann*, on post-mortem evidence, to an interstitial lymphangitis: the lymphatics being found packed with inflammatory exudate or pus cells. *Assmann* contrasts this with the "meshwork" appearance which appears during a resolving lobar pneumonia, which is due, in all probability, to peribronchial alveolar atelectasis. This, unlike lymphangitis, rapidly disappears.

FRIEDLÄNDER PNEUMONIA

Infection with the Friedländer bacillus causes a pneumonia with a marked tendency to abscess formation. It may have an acute or insidious onset. Diagnosis depends upon the discovery of the *b. mucosus capsulatus* in the sputum or blood.

In the acute type, which clinically resembles acute lobar pneumonia, resolution does not occur and a septic course follows. The insidious type runs a protracted course and often results in a chronic infection. It may be mistaken for tuberculosis.

The consolidation is usually bronchopneumonic in type, and tends to be bilateral. Pseudolobar involvement, by confluence, often occurs. After a time destruction occurs within the consolidated areas, and abscess cavities are found. Most cases are fatal, but some survive and either resolve completely or heal by fibrosis.

RADIOLOGICALLY four stages are described by *Kornblum*.



(1) Bronchopneumonic, with larger patches than is usual in bronchopneumonia with peripheral distribution. (2) Coalescence and pseudolobar consolidation. (3) Rapid lung destruction often involving large areas. (4) Resolution and fibrosis.

A *Fuso-spirochætal* Infection may produce a similar clinical and radiological picture. It may also resemble bronchopneumonia, lobar pneumonia, epidemic influenzal pneumonia, primary lung abscess, or acute caseous tuberculosis, or, in the more chronic form, may closely simulate chronic pulmonary tuberculosis.

CHRONIC INTERSTITIAL PNEUMONIA, FIBROSIS OF THE LUNG

The organisation of lobar or bronchopneumonia is sometimes referred to as chronic interstitial pneumonia. In these conditions the process is really one of cicatrisation—a reparative process, in which the fibrous tissue is often laid down in excess, so that a whole lung, or a large part of a lung, may become shrunken and indurated. A similar process occurs in the healing of infarct, abscess, gangrene, or tuberculous disease, and as a result of excessive irradiation.

Widespread fibrosis of the lung is most commonly seen as a result of an extensive influenzal or non-specific pneumonia or of tuberculous disease, involving the larger septa, and is usually unilateral. A progressive fibrosis of the fine interalveolar and peribronchial connective tissue, to which some authorities would confine the term “interstitial pneumonia” (carnification of the lung), occurs in chronic passive hyperæmia, associated with mitral stenosis and in pneumoconiosis. These are always

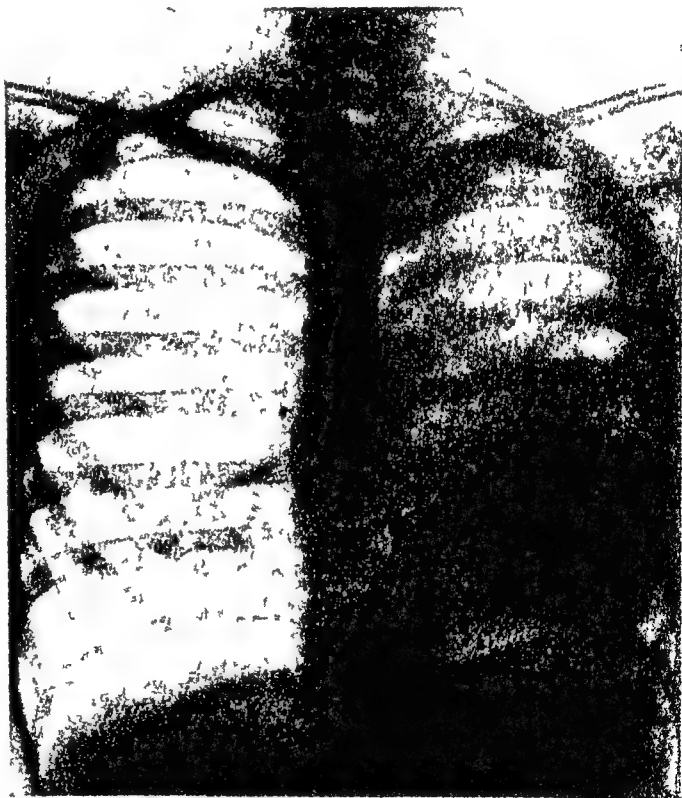


FIG. 162.—Pulmonary fibrosis. The heart is displaced entirely to the left of the midline. F. 13. No recent history of pneumonia. Clinical signs of bronchiectasis.

bilateral, and there is less tendency for the lung to shrink than in the reparative forms of fibrosis. Interstitial pneumonia in the acute stage follows the infectious pneumoniae, and may also arise in suppurative lesions of the pleura, mediastinum, neck, or vertebrae. The process extends along the peribronchial and vascular lymphatics and interalveolar tissues, i.e. the supporting stroma of the lung, in the form of white lines of several millimetres in diameter, outwards from the hilum, or inwards from the pleura (pleurogenous pneumonia). The bronchi may be obstructed and areas of atelectasis occur: blocking of the finer bronchi by fibrous tissue leads to bronchiolitis obliterans.

In the radiogram these lines may be visible, but are usually masked by the concomitant bronchopneumonia or atelectasis. During the chronic stage the radiological appearances of fibrosis of the lungs vary with the extent and type. Two signs are to be looked for: the shadows of fibrous streaks in the lung, most often seen in cicatrising tuberculosis, and evidence of traction. It is upon the latter sign that the diagnosis usually depends. Traction may produce the following signs:

(1) A falling in of the lung apex, often with crenation of the lung margin and thickening of the overlying pleura. (2) Displacement of the mediastinum and heart. (3) Displacement and angulation of the trachea. (4) Splaying out and distortion of the arborisations of the lung vessels. (5) Displacement of the diaphragm upwards with alteration of its contour, tenting, and adhesion. (6) Diminution of size of the affected hemithorax, closer spacing, and increased obliquity of the ribs.

Increased density of the affected lung or lobe is usually present, with compensatory emphysema elsewhere. Complete unilateral obscurity of one lung field may result from extensive fibrosis of a lung. The distinction cannot always be made from total atelectasis resulting from bronchial carcinoma. As a rule, in fibrosis, the opacity is less absolute and the displacement of the heart more marked. It is sometimes pulled into contact with the lateral chest wall. Nodular forms of lung fibrosis are described under pneumoconiosis.

Fibrosis following Irradiation

Irradiation fibrosis has been described by *Groover, Christie and Merritt*, by *Evans and Leucutia*, *Desjardens, Finzi*, and *Downs*. Post-irradiation fibrosis is not necessarily permanent, as *Holfelder* has demonstrated in the case of a patient cured of a sarcomatous metastasis in the lung by deep therapy. A large area of indurated lung gradually cleared up over a period of a few years. Seven and a half years after treatment the lungs were radiologically normal. *Downs* considers that there are two distinct types of lesion found after irradiation for carcinoma of the breast: (1) *Transient lung changes*. These are not due to fibrosis, but to pleuro-pulmonitis. A hazy, sometimes patchy, density extends out from

the hilum to the periphery; it may appear eight weeks after an intensive treatment, but more often follows re-treatment. It begins to fade after a few months and disappears at the end of a year. It is probably due to oedema.

(2) *Permanent lung changes.* On the evidence from post-mortem in seventy

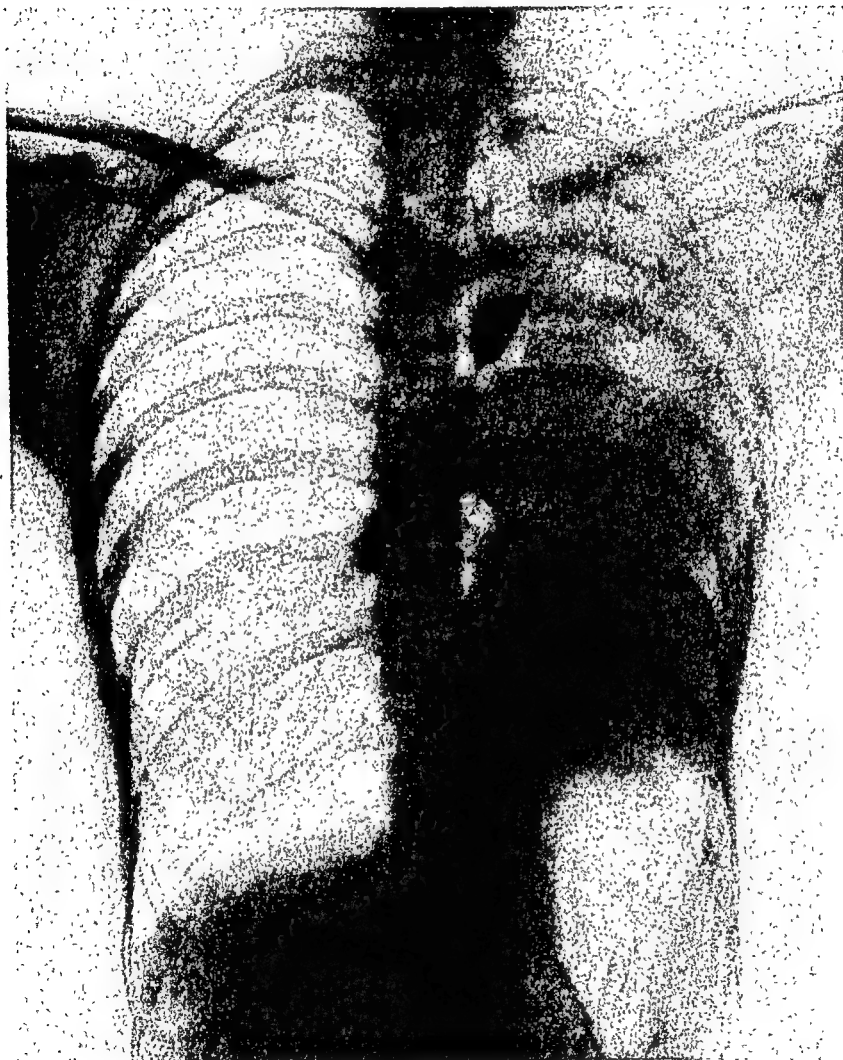


FIG. 163.—Fibrosis of lung, with displacement and narrowing of œsophagus. Dysphagia.

cases of cancer of the breast, of which fifty-three had been irradiated, *Downs* concluded that permanent fibrosis due to irradiation is very rare. Only one case was found in which he attributed it to the treatment. This case had been treated by radium only. Fibrosis rarely occurs unless the lung is previously damaged by metastasis, chronic disease, or infection.

Lung Fibrosis due to Mineral Oils

Intratracheal injections of mineral oils, or long-continued use of nasal medication with such oils, may lead to changes in the lung due directly to the oil which has reached the bronchi. These are shown radiologically as follows:

(a) In the early stages a miliary mottling of the affected area, with accentuation of the finer lung markings, resembling a fibrous bronchiolitis obliterans; oil droplets are found in the sputum.

(b) Later, progressive solidification and contraction of the affected lobe; at autopsy, a fibrosis of the alveolar walls was found in a case reported by K. S. Davis. The changes are usually more marked in the right lung. Such changes do not occur after lipiodol injection, and vegetable oils appear to be innocuous.

CHAPTER XXIII

LUNG ABSCESS

LUNG ABSCESS is a suppurative focus commencing in the lung parenchyma, and may arise from a variety of causes :

(1) POST-PNEUMONIC.—This form probably results from a mixed infection of pneumococci and streptococci. It may occur in lobar pneumonia or broncho-pneumonia. In the latter case the abscesses are often small and multiple. The onset and course are usually acute in the post-pneumonic form, but the abscess may become chronic.

(2) FOREIGN BODIES.—An aspirated foreign body is very liable to lead eventually to suppuration in the atelectatic area supplied by the blocked bronchus : peas, beads, fragments of tooth, tonsil or adenoid vegetation are common causes. The bronchial obstruction is due in part to inflammation of the bronchial mucosa and to retained secretion.

(3) PYÆMIC ABSCESS.—Infected emboli may reach the lungs in puerperal sepsis, osteomyelitis, intracranial sinus thrombosis, and many other septic conditions, through the systemic veins. They are usually small and multiple.

(4) POST-OPERATIVE.—It is disputed whether cases of abscess in the lung following operation are due to inhalation of septic matter during anaesthesia or whether they are embolic. The inhalation of blood, saliva, vomit, or mucus has been held responsible ; in the United States many cases have followed tonsillectomy. Lord found that out of 227 cases seventy-eight followed operations on the upper respiratory passages, and twenty-one followed extraction of teeth. Local anaesthesia lessens the risk, and has been widely adopted for tonsillectomy in the United States for this reason (*Ogilvie*). Some believe that an embolic origin is more probable, since abscess may follow abdominal operation for septic conditions. The inhalation theory is supported by the fact that the anaerobic organisms and spirochaetes found in septic foci in the gums and teeth are often present in the lung abscess. Post-operative massive collapse is very liable to terminate in abscess formation unless relieved by postural or bronchoscopic drainage.

(5) BRONCHIECTATIC.—Pulmonary suppuration may occur as a complication of bronchiectasis in the form of multiple ragged foci of parenchymal suppuration in the diseased area.

(6) NEOPLASTIC.—Carcinoma of the lung is very prone to suppurate. It is said that suppuration, with necrosis of the tumour, occurs in at least 25 per cent. of cancers of the lung.

(7) OTHER CAUSES.—Abscess may also result from penetrating wounds of

the thorax, from extension of subphrenic abscess, or of œsophageal carcinoma, or from the infection of a hydatid cyst.

DISTRIBUTION OF ABSCESS.—There seems to be a definite predilection for the right lung. *Lockwood*, in a review of the literature, found that abscess occurred three times as often on the right side as on the left, and twice as often in the lower lobe as in the upper. In 75 per cent. of cases the abscess is situated in the periphery of the lung and involves the pleura.

Localisation.—The abscess is, as a rule, localised to one of the bronchopulmonary segments (Figs. 104–106). Though this does not entirely prove the theory of bronchogenic origin, it is in favour of it. *Glass* has emphasised this localisation in bronchopulmonary-segments. The corresponding bronchi are readily visualised by bronchoscopy, when pus may be seen exuding from them (*Pinchin* and *Morlock*). Abscesses occupying the inner lung field in the postero-anterior view are almost invariably in posterior segments (i.e. in the distribution of the dorsal branches of upper, or more frequently lower, lobes, and are therefore paravertebral. A possible exception is an abscess in the segment supplied by the retrocardiac bronchus which lies close to the heart, at mid-depth in the lower field. To this exception must be added the occurrence of abscess in the middle lobe, which may be seen in the inner lung field, and the rare possibility of an abscess in the lingual tip of the left lung, which the writer has never actually met with.

The apex of the lower lobe is quite often the site of an abscess, and if the segment supplied by the outer branch of its bronchus is involved, the shadow in the postero-anterior view will lie towards the outer lung field. Usually it is in the mid-field—"parahilar" in the postero-anterior view.

Abscesses in the segments supplied by the axillary branches of the upper lobe and lower lobe bronchi lie towards the lateral chest wall. The remainder occupy the mid-field. These may be anterior or posterior, and it is therefore imperative that careful lateral and oblique views be taken in every case, in order to obtain precise localisation. If surgical operation is contemplated, it is also wise to supplement the usual erect views by views taken with the patient lying, especially in the case of basal lesions; owing to the change in position of the diaphragm, the lesion comes to lie higher in the chest with the patient recumbent, and this film will be a better guide to the surgeon than one taken with the patient erect.

The Radiological Appearances are at first those of pneumonia or pneumonitis of a more or less limited area of lung, with later an air-containing cavity and fluid-level within the shadow: to a great extent they will depend upon the type and stage of the abscess. *Pinchin* and *Morlock* suggest the following clinical divisions:

(1) Pneumonitis, preceding abscess formation.

(2) Pyogenic type: (a) Simple, with or without localising reaction. (b) Gangrenous liquefactive type.

(3) Putrid or spirochaetal type : (a) Secondary to simple. (b) With local reaction. (c) Fulminating gangrenous.

(4) Multiple. Either (a) Simple pyogenic, or (b) Gangrenous.

PRE-ABSCESS STAGE OR PNEUMONITIS.—This shows a diffuse homogeneous shadow, with ill-defined edges. Its size and shape will depend on several



FIG. 164.—Specimen : Lung abscess. The apex of the left lower lobe, supplied by the first dorsal bronchus, is consolidated and necrotic. This is a fairly common site. An abscess in this region may simulate a " hilar " cavity in the postero-anterior radiogram.

factors. If it is metapneumonic, it will show as a patch of persistent opacity in a lung field showing evidence of resolution elsewhere. If there is a blocked bronchus, signs of atelectasis (interlobar shift, concavity of margins) must be expected. If the abscess has arisen *de novo*, the patch of pneumonitis tends to be rounded in form, an expression of a rapid centrifugal spread of infection from an initially small focus. Many of these rounded shadows are found, however, on examination in several planes, to be " end-on " views of a pyramidal or cone-shaped lesion corresponding to a bronchopulmonary segment, the base of which may reach the pleural surface.

Pyogenic Abscess.—There is really nothing to distinguish the pneumonitis of the early stage of abscess from any other pneumonitis, but repeated views at

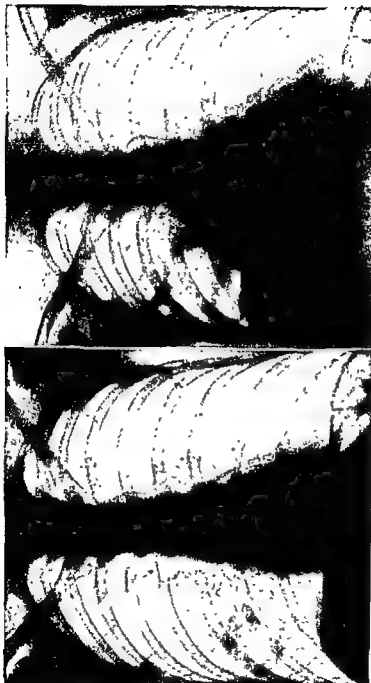


FIG. 105.—Abscess in right lung—stage of consolidation (pneumonitis).

Same case nine days later. Rupture into pleural cavity: pyopneumothorax.

short intervals are necessary to detect the first specific sign of abscess formation—an air bubble. Liquefaction of the area of pneumonitis usually progresses rapidly, and the occurrence of discharge into a bronchus soon enables a diagnosis to be established. The central cavity may be single, or consist of several chambers or loculi, usually communicating with a central space.

Tomography is valuable in bringing to light these commencing cavities,

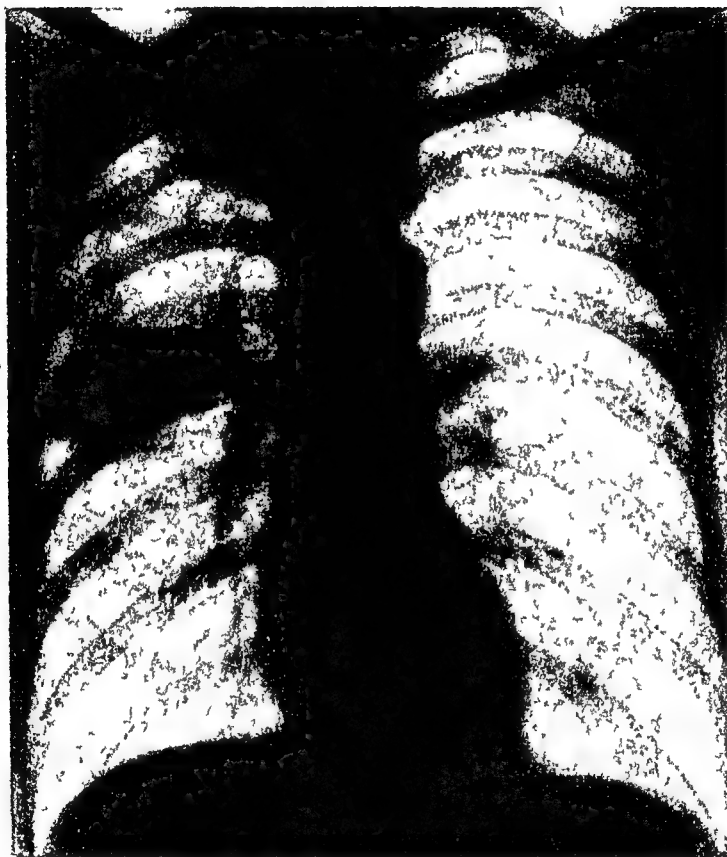


FIG. 166 —Abscess of lung, in axillary segment of right upper lobe.

or centres surrounded by large areas of consolidation. It may also show the drainage bronchus clearly.

After this has occurred, simple abscess may follow one of several courses :

- (a) The cavity may quickly close, and the area of pneumonitis disappears.
- (b) The small central cavity may increase in size till it reaches the limits of the original area of pneumonitis, showing a fluid-level which extends the whole width of the original shadow (liquefactive, or simple gangrenous type).
- (c) The cavity may remain surrounded by a ring-shaped protective barrier of condensed tissue, giving a thick-walled ring shadow in the radiogram.



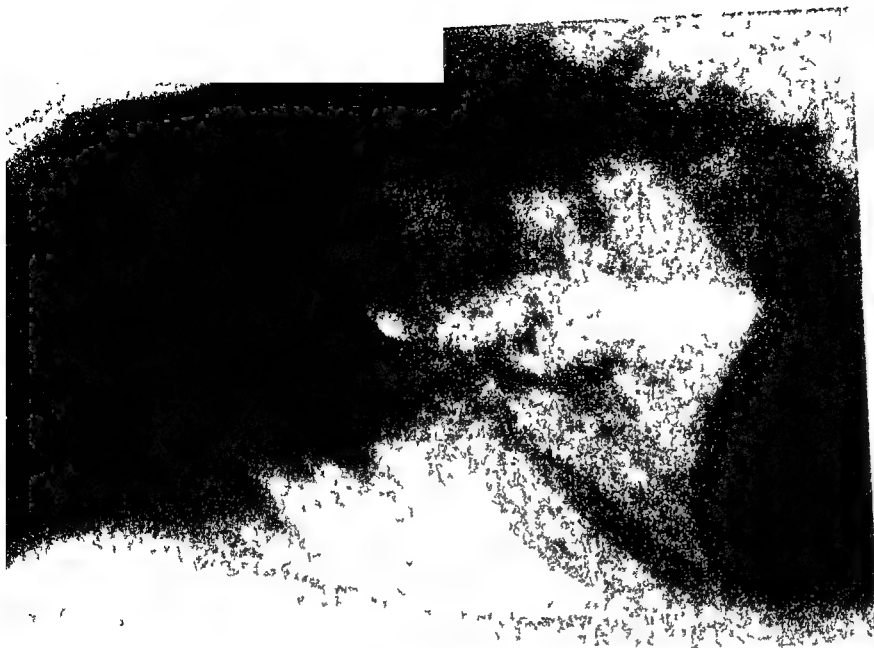
FIG. 107.—Abscess of lung: plain radiogram shows an area of pneumonia; the cavity is not visible.



Tomogram of same case in a plane $2\frac{1}{2}$ inches from front of chest. Abscess cavity clearly shown: its walls sharply defined: it is divided by incomplete septa.



Fig. 168.—Abscess of lung, multiple, following tooth extraction
Pneumonitis right upper lob. Cavity apex of left lower lobe.



Lateral view. Wedge-shaped area of pneumonitis in
right upper lobe posterior apical and axillary segments.

THE FUSOSPIROCHÆTAL OR PUTRID TYPE, if primary, breaks down rapidly, and the patient's condition is usually bad. If the spirochætal infection supervenes on a pyogenic abscess, the primary localising reaction tends to limit the rate of spread and to mitigate the severity of the symptoms.

MULTIPLE ABSCESSES may result (a) from bronchopneumonia (Friedländer bacillus); (b) from inhalation of many septic particles; (c) from bronchogenic spread from an existing abscess; (d) from pyæmia; and (e) from bronchiectasis with secondary infection. The Friedländer abscess has a tendency to rapid extension, and may quickly destroy large areas of lung.

The radiological picture of an air-containing cavity with a fluid-level which shifts with change of posture within an area of pneumonitis is usually, in the light of the history and signs, distinctive, but if the involved area is small it may be missed. If the evacuation of the liquefying core of the abscess is only just commencing, the cavity and fluid-level may be hidden by the surrounding opacity. Oblique illumination of the film may assist in the contrast sufficiently to show it. A fluid-level in an abscess situated posteriorly in the para-vertebral region may be confused with the outline of the vertebral body: by inclining the patient forwards this source of error may be eliminated. A fluid-level in the posterior recess may be hidden by the diaphragm. An antero-posterior or postero-anterior view with the trunk inclined forward will then bring it into view. Owing to the rapid course, cases of suspected abscesses require to be examined almost from day to day.

LIPIODOL IN LUNG ABSCESS.—Lipiodol will often fail to enter a lung abscess owing to blocking of the bronchus or the filling of the cavity by pus. During the stage of pneumonitis a typical appearance is shown after lipiodol injection, the area of pneumonitis being distinguished as an opacity in which no filling of the smaller bronchi or of the alveoli occurs. The bronchi leading to this region have few fine branches, and resemble the bare limbs of a dead tree.

During the stage of pneumonitis the abscess cavity and the alveoli in the pneumonic area do not outline with lipiodol. After rupture of the abscess into a bronchus the pneumonitis subsides and the alveoli in the pneumonic area can be filled with lipiodol. The cavity of the abscess, at first irregular, multiloculated and difficult to outline, becomes better defined in the course of time. A cylindrical dilatation of the drainage bronchus may often be observed: bronchiectasis in the form of cylindrical or ampullary dilatations of the bronchi in the affected segment of lung may occur as a result of fibrosis. Basal bronchiectasis is a common accompaniment of upper lobe abscess (Farinas).

Differential Diagnosis.—In the differential diagnosis of abscess of the lung the following conditions should be considered:

(1) Tuberculous cavities; (2) Cavernous neoplasm, or, during the pre-liquefactive stage, solid tumour; (3) Interlobar effusion with rupture into a bronchus (interlobar pyopneumothorax); (4) Congenital cyst of the lung;



FIG. 169.—Lung abscess right upper axillary segment.

Lipiodol injection in the same case. Lipiodol fails to enter the affected segment.



FIG. 170.—Abscess in right middle lobe



Lateral view. The middle lobe is consolidated and contains a cavity with fluid level; the position of the air bubble excludes interlobar empyema.

bullous emphysema, and acute vesicular emphysema ; (6) Bronchiectasis ; Actinomycosis and other mycotic infections ; (8) Pyopneumothorax.

Tuberculous Cavities.—An abscess in the upper lobe may exactly resemble an early tuberculous cavity. In both instances the radiological appearance would show a thin-walled ring with fluid-level. It is well known that a cavicular tuberculous focus may rapidly break down and disappear without trace. The same thing may occur with abscess, and in both conditions the clinical symptoms may be slight : in abscess the onset tends to be more abrupt, the course more acute, the evolution more rapid. The only real distinguishing feature is the sputum.

Cavernous neoplasm may show a ring shadow exactly resembling an abscess cavity. A point in the differentiation is the thicker wall of the neoplasm, with centrally placed cavity. In abscess the air bubble quickly tends to rise to the top of the consolidated area. The wall of a cavernous neoplasm may show nodules of growth projecting into the cavity, the outline of which in this case is irregular. This feature is by no means constant. Cavernous neoplasms occur of all intermediate types between a solid mass with minute cavities, and a thin-walled ring shadow with a fluid-level extending right across. All depends upon the completeness with which the neoplasm liquefies. A cavernous neoplasm is, in effect, an abscess. Bronchoscopy may help, but if the origin of the neoplasm was in a small bronchus or in a lung parenchyma, it may fail to make the diagnosis. The history, the character of the sputum, enlargement of hilar or tracheobronchial glands, or enlarged cervical glands, the presence of secondary lung nodules or of a primary carcinoma elsewhere in the body, and rib involvement, will afford collateral evidence.

Congenital cystic disease of the lung usually shows multiple translucent areas separated by thin septa, and fluid-levels are often absent or small in amount. A solitary congenital cyst may, however, resemble abscess, and the history and clinical condition must decide the issue. The long course and absence of acute symptoms or of foul sputum are the main distinguishing features.

An emphysematous bulla might on rare occasions be mistaken for abscess, but the clinical picture would be decisive.

A hydatid cyst or an interlobar empyema which has opened into a bronchus may produce momentarily an X-ray picture simulating abscess.

Bronchiectasis is distinguished by multiplicity of the cavities. A single bronchiectatic cavity may occur in the hilar region with little surrounding reaction. A tuberculous cavity may occupy the same site.

Actinomycosis or other mycoses may present the radiological appearances of a chronic abscess. Repeated sputum investigations and medication with iodides may clear up the diagnosis.

The tendency for pulmonary suppuration, as well as pulmonary neoplasm, to metastasise to the brain should be remembered. Examination will sometimes throw light upon cases showing obscure cerebral

CHAPTER XXIV

PNEUMOCONIOSIS

PNEUMOCONIOSIS DENOTES fibrosis in the lung, resulting from the inhalation of dust. The serious forms of disease are almost invariably due to the presence of some form of silica. The most dangerous pneumoconioses are silicosis, produced by dust containing silicon dioxide, and asbestosis, in which the active constituent is magnesium silicate. The inhalation of coal-dust, anthracosis, is a benign condition.

SILICOSIS

It was formerly held that free silica (SiO_2) was the only active agent in producing fibrosis of the lungs, which is the essential pathological condition. *Heffernan* and *Green* believe that the disease results from the local action of colloiddally transformed silica on the lung tissue. *W. R. Jones* believes that under mining conditions the agent is sericite, the hydrated silicate of aluminium, and not the silica-containing quartz.

Pathological Stages

(1) Entry of dust into the alveoli. Most of the particles found in the lung are from .5 to 5 microns in size. Particles over 10 microns in size are innocuous, since they cannot be phagocyted.

(2) Dust, having entered the alveoli, sets up a catarrhal reaction and cell proliferation. The proliferated cells become phagocytic to the dust.

(3) These "dust cells" enter the lymphatics and are carried to lymphoid deposits of the lobule, where some remain, others passing farther along the lymphatics of the lung. *Miller* has demonstrated the arrangement and grouping of the lymphoid deposits of the lobule of the lung, and the points at which the first deposits of dust cells occur. These are indicated in Fig. 171. In addition to these lobular deposits, others occur farther along the lymphatics near the point of bifurcation of the bronchi and vessels.

(4) Local stasis and cell proliferation at this point lead to the formation of pseudo-tubercles (*Mavrogordato*).

(5) Around these tubercles silicotic fibrosis develops which determines the radiological findings, this fibrosis being due to the activities of fibroblasts. The changes begin when the particles of silica or silicate contained by the phagocytes, interacting with body fluids, are converted into hydrated colloidal silica which, acts upon the protoplasm of the dust cells and then on the sur-

rounding cells. A fibrous wall is formed around these and around the central mass composed of silica sol (SiOH_4) and dissolved protoplasm. The colloidal silica eventually becomes dehydrated and inert, but so long as unconverted pure silica or silicates remain, silicosis is progressive. The rate of progression depends upon the speed of formation of fresh colloidal silica, which is accelerated

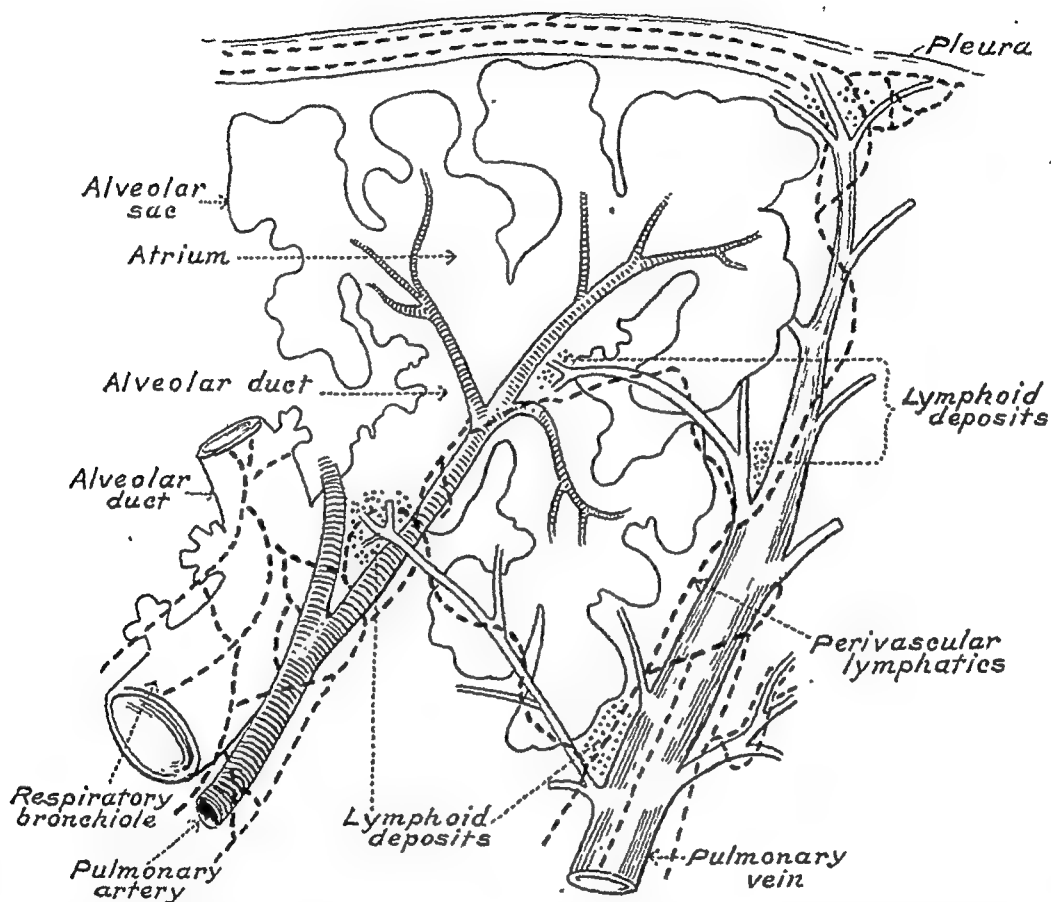


FIG. 171.—Diagram: Primary lobule of lung. The course of the lymphatics is indicated by a broken line. Lymphoid deposits dotted. (After W. S. Miller.) The respiratory bronchiole divides into two alveolar ducts bearing atria, each of which has a number of alveolar sacs, on the periphery of which are the alveoli. A branch of the pulmonary artery goes to each atrium and is distributed to the alveolar sacs. The pulmonary vein is situated between the primary lobules.

by the presence of alkalis. For this reason scrubbing powders which contain alkali are dangerous and lead to rapid development of silicosis. The pseudotubercles are microscopic, but when fibrosis has reached a certain stage around them, the silicotic nodules become visible in X-ray. This may be preceded by what has been called the first visible stage of silicosis, namely, a diffuse veiling of the lung field with some increase in density of the pulmonary stroma shadows.

and in the richness of their arborisation. This depends upon the rapidity of the accumulation of dust cells and the degree of lymph stasis and clogging of the lymphatics. Some of the cells may pass through the lymphatics and set up fibrotic changes in the interstitial lung tissue.

There are three points at which the dust carried by the phagocytic dust cells may accumulate in the lung :

(1) *In the peribronchial and perivascular lymph vessels.* When these are blocked, there is an increase in the density and richness of arborisation of the lung markings. This peribronchial type occurs, according to *Pancoast* and *Pendergrass*, among the coal-mine workers who break stones ; they develop it very slowly.

(2) *In the lymphoid deposits in the lobules, at the bronchial bifurcations, and the hilar lymphatic glands.* Accumulation of dust cells at these sites produces the nodular form of silicosis. This is much the most common form in stone-masons, pottery workers in pure flint processes, miners working in hard rock, sand-blasters, and metal grinders using sandstone wheels.

(3) *In the interstitial tissue of the lung*, that is, in the interlobular and alveolar septa. This type results from a rapid and complete obstruction of the lymphatic pathways, with egression of the dust particles from them. This type, without nodular changes, is said to occur in granite cutters ; workers in scrubbing-powder factories also develop this diffuse form.

It must be emphasised that the rate of intake of active dusts has a profound effect upon the radiological appearances in the early stage. It seems certain that in some trades there are definite early prenodular radiological findings, and in other trades in which intake is slower and elimination to some extent keeps pace with it, the early phenomena may be entirely absent and the radiological manifestations begin with definite nodulation in the lung periphery. The end stage is one of diffuse dense fibrosis which dominates the picture. If nodulation has been prominent, the nodules then become confluent and form large irregular masses throughout the lungs. There may, in addition, be an extensive diffuse fibrosis or carnification in the lung, as a late result of blocking of the lymphatics. Pleural thickening and adhesions are almost invariably present at this stage.

The trades in which the risk of pneumoconiosis arises are very numerous, and a full review with extensive literature was given by *Pancoast* and *Pendergrass*. Miners, hard-rock drillers, pottery workers, sand-blasters, metal grinders using sandstone wheels, stone-masons, workers in silica brick manufacture, persons engaged in the manufacture of scrubbing powders and in many other trades are liable to contract the disease. Sand-blasting is one of the most serious risks. *Meiklejohn* found that of the sixty fatal cases of silicosis occurring in the years 1929-33, with exposure of under fifteen years, thirty were sand-blasters. The incidence in the pottery industry and in sandstone workers is described by *Sutherland* and *Bryson*. *Møller* found positive radio-

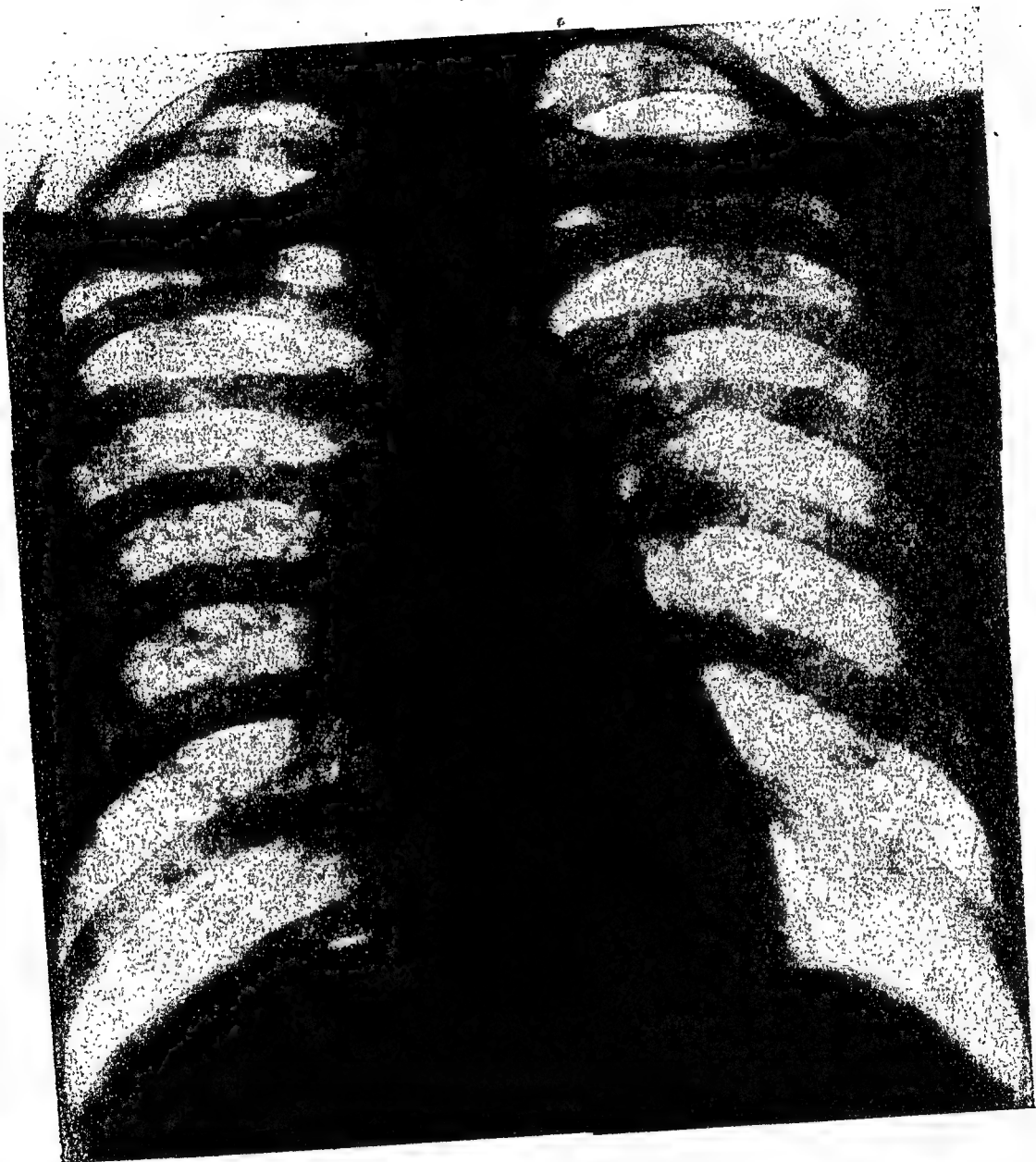


FIG. 172.—Silicosis (stone-mason).
Multiple disseminate nodular fibrosis.

logical evidence in 45 per cent. of 798 pottery workers, including incipient cases. Exposure to the dust of powdered flint, in bedding china-ware in flint preparatory to firing, is one of the chief sources of silicosis in pottery workers. It is possible that this material will in the future be replaced by alumina; a



FIG 173 —Pneumoconiosis (stone mason).

preliminary survey of operatives exposed for many years to alumina dust has shown no clinical or radiological evidence of occupational fibrosis of the lungs in these workers. The rate of development of the disease in the different industries is very variable, and depends upon the degree of exposure and the amount of silica inhaled. In the South Wales mines, for example, rock drillers working almost constantly on rocks with a high percentage of silica have

a high incidence. In 1936, 268 certificates of disablement were issued in this area. One hundred and eighty-two of these were in anthracite miners.

Bromley has emphasised from his study of silicosis in pottery workers that each occupational risk, even in the same industry, must be considered individually, since exposure must vary widely with the nature and condition of the work. In this risk he has drawn attention to a "mixed dust" picture occurring principally among workers in earthenware body, which he regards as characteristic.

Lyle Cummins distinguishes between chemically active (silica, asbestos) and chemically inert dusts (e.g. coal-dust). The former are soluble in body fluids and cause lesions of two types: toxic, leading to local cell-death, and sclerotic, leading to nodular or diffuse fibrosis (silicosis, asbestosis). The chemically inert dusts are insoluble in body fluids, but may, if accumulated to any marked extent, exert a mechanical "foreign body" action and cause fibrosis around the dust deposits. These inert dusts in pure form produce little fibrosis; the



FIG. 174.—Pneumoconiosis, acute. Patient, a man of 47, had worked in a "dry soap" factory for 4½ years. No previous exposure to silica. Death four months later. Autopsy findings: A small tuberculous cavity in right apex. No tubercle in left lung. The visible changes are due to silicotic fibrosis.

lungs are well aerated, but stained with pigment in coal-trimmers. X-ray examination shows no changes, as a rule, or at the most partial small mottling. But if there is a lymph stasis, due to a previous or simultaneous inhalation of active dust, or to some other lesion, such as tuberculosis, *Cummins* believes that the inert dusts may accumulate to a dangerous extent and produce areas of diffuse fibrosis (silico-anthraxis, silico-siderosis). This may occur, for example, in coal-miners who work from time to time in hard rocks, or are exposed to stone dust produced by pneumatic drills or by blasting.

Classification of Silicosis

Three stages are recognised in this country for the purposes of Workmen's Compensation (Silicosis) Act of 1928. The compensation under this Act is payable for disability caused, not on the fact that silicosis is present. The stages are :

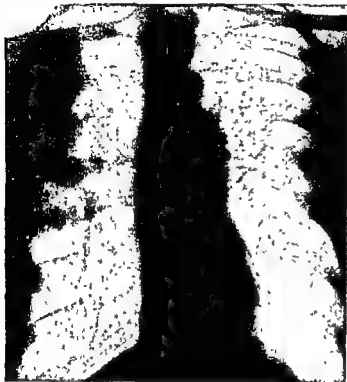


FIG 175 —Pneumoconiosis M 47 Gunster worker for 16 years
Diffuse type of fibrosis

FIRST STAGE, characterised by (a) the appearance of the earliest detectable physical signs of the disease with (b) radiological evidence not less than *nodular shadows* together with an increase of the hilum shadows and pulmonary reticulum, and (c) with or without impairment of capacity for work.

SECOND STAGE, characterised by (a) further development of the physical signs found in the first stage ; (b) an increase in the area of nodular shadows, with a tendency to confluence of the individual nodes, and the pres-

ence of symptoms with some degree of impairment of capacity.

THIRD STAGE, signs, symptoms, and disability in excess of Stage II. In this stage there are often found massive confluent shadows, and gross pleural changes.

Radiological Classification

The above classification is adequate for working purposes. Very many classifications of pure radiological appearances have been suggested, based on a grading of large numbers of films reviewed. They are all, in the writer's opinion, useful only when a study of a large number of films is being made in some particular industry or investigation. Such a classification as the following is meaningless apart from its context :

(1) Rather more fibrosis than usual; (2) More fibrosis than usual; (3) More fibrosis full; (4) More fibrosis to commencing fibrosis; (5) Commencing fibrosis plus; (6) Commencing to early; (7) Early commencing; (8) Early to medium; (9) Medium fibrosis; (10) Medium to advanced fibrosis; (11) Advanced fibrosis (*Brink, cit. Pancoast and Pendergrass*).

A classification suggested by *Pancoast* and *Pendergrass*, based on a combination of radiological and pathological features, is helpful, but, cannot be strictly applied in every case in actual practice.

(1) Peribronchial perivascular lymph node predominance: (a) rapid; (b) slow.

(2) Early interstitial predominance (interferes with diaphragmatic movement): (a) with nodular appearance; (b) without nodular appearance, rapid or slow.

(3) Late or advanced interstitial predominance.

(4) Nodular predominance: (a) non-progressive; (b) progressive.

(5) Advanced diffuse or terminal fibrosis: (a) conglomerate nodular type; (b) interstitial type; (c) massive fibrosis type.

It is evident that in all stages preceding the formation of definite nodules the appearances met with are difficult both to describe and to classify. Since the working classification previously referred to begins with the appearance of nodules, as the first specific evidence of silicosis, the following account of X-ray appearances of the disease may conveniently commence at that point also. The prenodular manifestations, variously described by different observers, will be dealt with later.

It seems certain that the latter differ from trade to trade, and probably from one individual to another. It is certain that in many cases no clearly describable prenodular stage occurs at all; in others the radiological changes are unspecific, in that they are overlapped by "normal" appearances, i.e. they can be matched by films taken from samples of the adult population.

In any case, the stages are only to be considered as milestones along the march of the disease; or perhaps not so much "milestones"—which imply equal distances and equal rates of progress—as arbitrary stations at which it is convenient to collect and group individual silicotic patients. This does not mean that the rate of progress from one stage to another is uniform, or that all silicotic patients cover the same ground during the course of the disease. Some patients, indeed, die of their disease while presenting only mild radiological signs; others with extensive involvement are capable of full work, while others again progress rapidly from early to terminal stages without passing through a typical second stage. *Reichmann* prefers to speak of a "mild" stage and a "severe" stage, in an endeavour to avoid the difficulties caused by the arbitrary selection of three stages. Compensation in this country is usually based, not upon the fact that the patient has silicosis, or presents a type of X-ray picture, but on the degree of disability resulting from the

Radiological Features

IN THE FIRST STAGE, as already defined above, the nodules are radiographically dense and circumscribed, owing to the surrounding emphysema. Occasionally they calcify, and then become more opaque, though they rarely show visible calcium granules in an X-ray film. The nodules are first seen along the axillary border of the upper lobes; later they tend to be massed in the lateral and posterior parts of the upper lobes, and in the apex of the lower lobes. In the more acute cases, where the intake of dust is rapid, the lymphatics become quickly clogged and the consequent peribronchial and perilymphatic fibrosis leads to an element of diffuse shadowing, which cannot be analysed into component parts. This has been found chiefly in sand-rock drillers, but is also described by *Flemming Møller* in pottery workers in the early stages of the disease.

The nodules when first visible vary in size from a pinhead to a pea. they gradually become denser and larger, and in long-continued cases may calcify. They rarely show visible granules of calcium, but become extremely dense and sharply defined when calcified.

The site of first development of the nodules is variously described. They are said to appear (1) "round the root of the lung on the right side" (*Pancoast and Pendergrass*); (2) "in the upper lung fields between the clavicles and lung-roots" (*Entin, Klehmet, Kastle, cit. Assmann*); (3) "in the lateral mid-fields" (*Krause and Loben*), (4) "in the lateral middle regions"; (5) "in the lower lobe" (*Staub Otiker*).

The writer's experience, based upon the examination of a large number of silicotics during the past nine years, principally stone-cutters (bankerhands), leads to the following conclusions:

There are two areas in which the nodules tend to appear early and to be massed most thickly later on in the course of the disease.

(1) The postero-lateral region of the upper lobe.

(2) The apex of the lower lobe.

In the stone-masons, when only few nodules were present, these were invariably on the outer wall in the infraclavicular region of the upper lobe, and few if any were visible at that stage at the apices, at the bases or near the roots. A second focus of condensation was often found at hilum level, in the apex of lower lobe. When there were many nodules, they were found throughout the upper lobe, but more heavily massed at the outer and posterior parts except the extreme apices: they were very heavily massed in the apical regions of the lower lobe, as shown by a lateral view. The roots were comparatively free. This lower lobe apical grouping has been found also by *Pancoast and Pendergrass* and confirmed by autopsy findings. It is interesting to note this frequent involvement of the regions supplied by the posterior apical and axillary bronchi of the upper lobe and of the first dorsal bronchus of the

lower lobe in pneumoconiosis, as in some inflammatory diseases (tuberculosis, abscess).

In many published cases, particularly in pottery workers (*Bromley, Fleming, Moller*), the antero-posterior radiogram suggests that there is a predilection for the apices of the lower lobe. This gives rise to the rounded dense patches seen on either side of the hilum. *Bromley* states that this type of picture is common in the potteries in workers exposed to intensive silica risks.

The stage of nodulation is nearly always accompanied by emphysema. This possibly accounts for the infrequency with which one sees evidence of thickened roots and signs of diffuse fibrosis in this stage.

THE SECOND STAGE, as already defined, shows the nodules becoming larger, tending to coalesce into irregular opacities which gradually increase. Fibrosis becomes more extensive and contractions occur, most marked in the upper and posterior parts of the lung, which pull the trachea and mediastinum backwards, and may thus cause an apparent diminution in size of the hilar gland and even mask, by causing rotation of the heart, an enlargement of that organ. The lateral view is valuable in demonstrating these points.

Clinically there is often a surprising absence of symptoms, until large areas of lung are involved. The patients sometimes complain of shortness of breath, and there is a tendency to acquire other respiratory diseases, e.g. coughs, colds, and bronchitis. In the milder cases (e.g. in the writer's series of bankerhands) there is little or no impairment of capacity for work. The more rapidly progressive lesions (e.g. in sand-blasters) may incapacitate in a few years. *Bromley* quotes a case of a dry-soap worker who died after four and a half years' exposure: *MacDonald* and others report fatal cases in packers of cleaning powder containing silica, after two and three-quarters and four and a quarter years. *Radiologically* the differential diagnosis is from miliary tuberculosis, in which the nodules are smaller and more evenly distributed, from tubercular bronchopneumonia and from the mycotic infections. Sporotrichosis may simulate silicosis closely. When confined to the upper lobe and calcified, the nodules may suggest old tuberculosis of the adult type, but the symmetrical involvement, the even size of the nodules, and the absence of tracheal displacement usually make the distinction easy. *Moller*, in discussing pottery silicosis, draws attention to the fine network of reticular shadows joining the nodules as a point against tuberculosis; it does not, however, occur in all nodular silicoses.

THE THIRD STAGE is characterised by diffuse fibrosis. Three types occur:

(1) The larger nodules of the second stage coalesce, or lie close together, with poorly aerated hazy lung between them. The diaphragm movements are restricted and evidence of pleural involvement is found (diaphragmatic adhesions, interlobar sclerosis).

(2) A diffuse fibrosis, consisting of a very heavy pattern of dense broad lung shadows with patches of confluence, with or without evident nodules.

(3) Rounded parahilar masses, an extension of the lower lobe apex involvement previously referred to. Diaphragmatic involvement is likely to be present.

The picture may show huge blocks of fibrous tissue in the middle of subapical zones, or extensive patchy fibrosis still retaining a suggestion of the former nodular distribution. Very large emphysematous bullæ may be seen. The pleura is thickened. The diaphragm is displaced downwards by basal emphysema or distorted by adhesions. Rupture of emphysematous bullæ may lead to spontaneous pneumothorax. Clinically the patients are usually sick, incapable of work, dyspnoic, and suffer from secondary heart failure consequent upon the extensive pulmonary disease.

The difficulty in the third stage is to exclude a coincident tuberculosis. *Kettle* suggested that the tuberculous element is always present when dense fibrous blocks predominate, but this is by no means proved. Cavitation points strongly to tuberculosis, but even this is not pathognomonic, since non-tuberculous cavities may in rare instances result from the cutting off of the blood supply to an area of the lung as a result of the fibrosis.

"MIXED DUST" RISK.—Workers in silicates (slate rock, containing 40 per cent. of silicate, and 2 per cent. free silica) and in earthenware give a radiological picture in which the snow-storm nodular appearance may fail to develop or be much modified by strands, or later by patches, of fibrosis. This fibrosis is diffuse from the early stages. Some tendency to nodule formation may be seen as the disease progresses, but the characteristic picture is that of multiple fibrous patches throughout the lung field.

"INCIPIENT" STAGE.—The changes described as occurring in earliest stage, prenodular silicosis, are (1) enlargement of the hilum shadows; (2) evidence of interstitial fibrosis, in the form of a diffuse haze in the lung field; (3) evidence of peribronchial perivascular fibrosis, in the form of thickening of the lung reticulum and increase in the amount of reticulum visible. It corresponds to the stage of lymphatic block, with escape of some dust cells into the interstitial tissues, and early fibroblastic reaction. The infrequency with which the writer has actually met with any of these conditions has made him sceptical about them, but a careful review of personal cases has shown a few examples of all three. *Brink* and *Riddell* have noted a diffuse parahilar haziness resembling that produced by a small female breast or strongly developed pectoralis muscle, but higher up. *Flemming Møller* has also detected these "pseudomammæ." *A. R. Smith* found the appearance in rock-drillers exposed to an intensive risk. In a few personal cases in stone-masons there was a definite enlargement of the hilar shadows, in which the pulmonary arterial shadows had not the usual clear-cut edges, but were obscured by a diffuse shadow fading off towards the lung field. In these cases there was a slight loss

of translucency in the middle region of the lung field, in the situation where the rounded aggregation of nodules are found in the second stage. The evidence of peribronchial perivascular fibrosis, namely a richer and coarser network of arborisation, did not occur in the writer's series in stone-masons, but *Flemming Moller* has found it in porcelain workers and has demonstrated it convincingly in his published radiograms. The fine sharply drawn reticular pattern of the normal lung becomes much denser and more abundant: details are visible right out to the lung periphery. The separate lines are woolly, and lack sharpness. This appearance is seen also in cases of asbestosis. None of these appearances can be considered typical or conclusive evidence of silicosis, as they may be simulated by passive congestion, chronic respiratory infection, or bronchiectasis. The writer has seen a case of mitral disease with back pressure lung in a tool-grinder working with carborundum wheels, which was wrongly diagnosed as "silicosis." This condition is easily recognised: the shadows of the enlarged vessels diminish evenly in size when traced outwards from the hilum.

ASBESTOSIS

Asbestos, a variety of hornblende, is a silicate of magnesium. In appearance the crude product is a soft semi-translucent rock, with a marked fibrous structure. This rock is crushed, and the fibres are carded, spun, and woven into fabric for brake-linings and for heat-insulation materials. The chief risk is in the carding process, in which the brittle elements, removed in the carding, are inhaled. Fine fragments of asbestos fibre are also inhaled, and form the nuclei of the curious "asbestos bodies" found in the lung on puncture or post-mortem (*Cooke, McDonald, Gloyne, Ellman*).

The workers develop a cirrhotic condition of the lungs. It affects the lower lobes principally, and is a diffuse interstitial fibrosis. Of the 363 workers examined by *Merewether*, 26.2 per cent. were affected. Evidence of asbestosis may be found after three and a half years' exposure to risk, though this is not usually present until after six years' exposure to asbestos.

Clinically dyspnoea is the prominent symptom—in early stages only accompanying effort, in the advanced cases brought on by the least exertion. An irritable cough may be present. Cachexia occurs in the late stages.

The writer has had an opportunity of studying during the last seven years over two hundred films of workers in asbestos. The majority of these, engaged in weaving or spinning, showed no outspoken radiological appearances, even after fifteen to twenty years, but positives were encountered in weavers, disintegrators, mixers, and carders of eight to thirty years' exposure. Of 211 cases, 12 were definite positives, with well-marked changes, 14 had slighter changes, visible in the X-ray, and 6 were "suspicious." *Wood, Sparks, and Glick* examined 80 employees in 1928 in London and found definite evidence of asbestosis in nearly all.

Radiological Features.—In the early stages the picture is one of very fine mottling at the bases, of which the component specks are soft and faint ("ground-glass appearance," *Burton Wood*). The peribronchial and perivascular fibrosis is revealed as an abundant fine reticulum of lung shadows



FIG. 176.—Asbestosis

in which the nodulation is scattered like dew drops on a cobweb. These nodules may reach considerable size, but are always soft and fuzzy at the edges, never hard and distinct like those of silicosis. They sometimes show a lobular and acinous grouping in rings and rosettes, like the lesions of productive

marked in the perihilar and basal regions. This mottling is soft in character and lacks the peripheral distribution, the sharp definition, and the density of the typical silicotic nodule. The interesting feature is the early onset of the radiological appearances, in some cases within a year of commencing work. It is possible that the mottling in these early stages may be due to staining by deposition of an iron compound, comparable to the local "siderosis" which may cause a persistent shadow after removal of an iron foreign body from a bronchus. Post-mortem examination of two cases, which had shown typical snowflake mottling in their radiograms and had died of abdominal cancer, showed no nodulation and no abnormal fibrosis in the lungs. One of these men had worked for forty years in the industry. The condition is therefore not a typical silicosis, though it would appear that this condition may also occur in this industry.

(1) The lower half or two thirds of the lung clouded by "ground-glass opacity," seen on close inspection to be due to innumerable fine striations and punctate stippling. The characteristic nodulation of silicosis was not seen.

(2) Evidence of pleural adhesions or thickening, such as obliteration of costo-phrenic angles, apical thickening.

(3) Outline of cardiac shadow shaggy.

(4) Occasionally enlargement of the pulmonary artery, due to back pressure and over-distension.

Burton Wood and Gloyne, 1931, found ten cases of tuberculosis in a series of fifty-seven cases of asbestosis; nine were females, mostly young, whose susceptibility to tuberculosis would be greater than that of the average worker in dusty trades. Bronchiectasis may occur, but is not distinguishable without lipiodol.

While agreeing that the lesions are first seen at the bases, and may be confined to them, the writer has noted cases in which the disease has spread later to the whole lung on both sides.

SIDEROSIS

Pulmonary Changes in Electric-arc Welders.—Doug and McLaughlin have recently described fine mottling in radiograms of the lungs of electric arc welders, due to the inhalation of fumes containing finely particulate iron oxide, with possibly a little silica. The X-ray appearances are different from those of silicosis and asbestosis. It is at present uncertain whether the lesion is a fibrosis or a chronic inflammatory change. Of sixteen cases examined, with histories of exposure of six to sixteen years, six had positive X-ray appearances. The patients were not ill, and there was no evidence of gross fibrosis in the chest. The diaphragm movements were unimpaired.

Acute Siderosis.—Appearances rather similar to the above were found by Bentzen (1934) in a man employed for two months on a machine used for pulverising steel. The radiograms showed the lungs to be studded with miliary infiltrations, most closely packed in the hilar regions. He ascribed them to multiple miliary pneumonic areas, due to irritation. They disappeared in nine months. He compares them with changes found in some anthracite miners, described by Wainwright and Nichols. These writers found, on histological examination of lungs of men who had ceased work in the mines for several years, apparent regression of the lung changes. It would seem that part of the radiographic changes in such cases is due to non-fibrotic elements—hyperæmia, stasis, œdema, or lobular atelectasis.

Lung Changes in Hæmatite Iron-ore Workers.—Fawcitt¹ has observed in a considerable number of workers in the hæmatite iron-ore mines in Furness and West Cumberland a snowflake mottling distributed throughout the lungs, most

¹ R. Fawcitt, personal communication.

Occasionally the outline is extremely sharply defined. An organised infarct may persist for a considerable time, gradually shrinking in size. Hæmoptysis usually occurs, but some infarcts are clinically silent.

Fig. 178 shows an infarct in a female patient of 41 suffering from chronic heart disease. She had marked cyanosis and dyspnœa. The radiogram showed a somewhat triangular shadow in the right upper lobe, proved at autopsy to be due to a large hæmorrhagic infarct. The post-mortem specimen is shown in Fig. 179. The heart was much hypertrophied and the mitral and aortic valves diseased. There was no hæmoptysis, and the infarct was not suspected clinically.

Septic Infarct usually results from embolism after operation or in abdominal disease. It may break down rapidly and form an abscess cavity (embolic abscess).

SYPHILIS OF THE LUNG

Lung syphilis is uncommon. Up to 1920. McIntyre (cit. Freedman) found forty-eight reported cases with autopsy findings. Three forms have been described: (a) Gummatous; (b) Interstitial fibrosing (*Assmann, Bergenhoff*); and (c) Diffuse syphilitic lobar pneumonia. The latter, except in infants (*pneumonia alba*), is of doubtful occurrence. Five diagnostic criteria have been laid down: (1) Absence of grave symptoms; (2) Sputum negative for tubercle bacilli; (3) Presence of other syphilitic stigmata; (4) Positive Wassermann; (5) Response to antisyphilitic treatment.



FIG. 179.—Hæmorrhagic infarct of right upper lobe. Autopsy specimen of case shown in Fig. 178.

Interstitial Type.—The interstitial type has been described by *Bergenhoff* and by *Assmann* as arising in the hilum and showing shadows radiating thence, due to infiltration of the lymph spaces, and spreading along lymph vessels in the peribronchial, perivascular, and interlobular connective tissue. This proliferative process is most marked in the perihilar region and in the lower



FIG. 180 —Syphilis of lungs. (Autopsy.)

lobes. There is a marked tendency to a subsequent fibrosis and shrinking; bronchiectasis may occur. *Bergenhoff* has observed a number of cases of this type. In the absence of post-mortem control, the diagnosis is a somewhat hazardous one, as the appearance can be simulated by other conditions.

Gummatous Type.—Gummata may be small miliary nodes, or large and tumour-like, occurring anywhere in the lung fields, usually in the right lower

and middle lobes. They are usually sharply defined and round, or, if surrounded by fibrosis, of irregular shape. They may cavitate centrally. It is obvious that they may be indistinguishable from neoplasm of the lung. Radiologically, the only distinguishing feature from a nodular carcinoma is the rate of growth, slow in the case of gummata, rapid in carcinoma. The history of long duration of illness is incompatible with malignant disease.

Still rarer forms of pulmonary syphilis are : (1) Gummatous ulceration of bronchial mucosa, followed by fibrotic bronchial stenosis (*Lenk*); (2) Pulmonary arteritis. It is believed by some writers that Ayerza's disease is a syphilitic manifestation (*Konstam* and *Turnbull*).

PULMONARY MYCOSES

The importance of this subject lies in the fact that many systemic mycoses occur which are clinically and pathologically similar to tuberculosis, and that they may produce changes in the lungs, ranging from bronchitis to massive pleuro-pneumonic lesions, which resemble tuberculosis radiologically and can only be differentiated from it by exhaustive bacterial and mycological study of the sputum.

The radiological aspects have been studied as a whole by *Fawcitt*, whose paper contains over eighty references to bronchomycoses affecting the lung. Reference should also be made to *Dodge's* work (1936), to *Castellani* (1928), to *Brumpt*, and to articles in the American journals, of which the most recent and most detailed is that by *Carier*.

It is still open to question to what extent pulmonary mycoses may affect the general population in this country. That the incidence has, in most cases, a geographical or occupational distribution seems certain. For example, coccidioidal granuloma, affecting the skin, lungs, and oropharynx, is almost confined to California. Blastomycosis is common in the middle-west regions of the United States, and all mycoses are more frequent in the tropics or sub-tropics. Moniliasis has been described by *Marett* as of frequent occurrence in the Channel Islands. Among occupational risks may be mentioned actinomycosis, in persons exposed to hay and grain; hay-dust disease (Bronchomycosis *Peniseciorum*, *Fawcitt*), in farm-hands working with mouldy hay infected with *Aspergillus*, *Penicillium*, and *Mucor* (*Munro-Campbell*, *Fawcitt*); and pigeon fanciers' disease (*Aspergillosis*), affecting those who feed pigeons by hand with grain.

Fungi are of almost universal distribution. They are lowly plants, consisting of mycelial threads (hyphæ) and spore-bearing apparatus. Most of the 100,000 known species are non-pathogenic to man. Familiar examples are *Mucor mucedo*, occurring on damp bread, in soil, and in horse-dung; *Penicillium*, on mouldy cheese and grain; *Aspergillus*, on decaying vegetable matter; *Oidium lactis*, on milk and cheese.

Castellani classifies the bronchial mycoses as : (1) Due to yeast-like fungi—*Monilia*, *Cryptococcus*, *Saccharomyces*, *Blastomycoides*, and *Endomyces*.

(2) Due to filamentous fungi—(a) Slender, e.g. *Nocardia*, *Anaeromyces*, and *Vibriothrix* ; (b) Larger, e.g. *Oidium* and *Hemispora* ; (c) With characteristic fructifications, e.g. *Aspergillus*, *Penicillium*, *Mucor*, *Rhizomucor*, *Sporotrichium*, etc.

Coccidioidal Granuloma.—The organism, *coccidioides immitis*, is distinct from blastomyces. It affects the skin or mucous membranes, and as a systemic disease produces granulomatous lesions which are always fatal. The pulmonary lesions are essentially granulomata, and in the radiogram cannot be clearly distinguished from tuberculosis ; they show, however, certain tendencies, which diverge from those of tuberculosis. These may be summed up as follows. (1) The mediastinal and hilar glands are often enlarged even in adults. (2) Miliary spread is very common, but produces a vague ground-glass appearance, in which the individual lesions are not as sharply defined or distinct as they are in miliary tuberculosis.

There is little or no evidence of spread from the upper lobes downwards, and cavitation is rare in the older lesions. Cavities, if present, have no fibrous wall. Fibrosis tends to be absent from the picture ; linear fibrosis and retractions of the hilum and mediastinum are not seen. Nodular and acinous lesions do not occur. A striking feature is the tendency to produce destructive lesions in bone and abscesses in the thoracic wall, which are exceedingly rare in pulmonary tuberculosis.

Blastomycosis.—The most characteristic lesion is a chronic spreading cutaneous infection, with abscess formation, ulceration, and thickening of the epidermis. The infection may, however, gain entrance through the respiratory tract, and establish a bronchopneumonic patch, followed by growth of the organism. The radiological appearances are even more like those of tuberculosis than in the case of coccidioidal disease. Nodular manifestations are common, either miliary or coarse. Cavities, blotchy shadows, due to bronchopneumonic spread, fibrosis, and well-organised pleural thickening and adhesions are described.

The disease is not often recognised in its early stages if confined to the lung. Particular interest, therefore, attaches to the case shown in Fig 181, described by *Dr. R. L. Rawlinson* :

M., æt. 29. History.—At the age of 19 attended *Guy's Hospital* suffering from bronchitis and loss of weight. Attended at *Brompton Hospital* in 1931 with cough, dyspnoea, and moderate expectoration. Clubbing of fingers, physical signs of bronchitis. *Blastomyces* isolated from the sputum. An X-ray examination in May, 1931, showed very heavy hilar shadows with evident adenopathy. The enlarged glands are in general not clearly separable from the hilar shadow, but a distinct nodular shadow is apparent in the left hilum just above the left branch of the pulmonary artery. In addition the

original film showed generalised small patches of mottling and fibrosis. The patient was treated with potassium iodide and improved greatly. A second film, taken in September 1934, showed marked improvement in the lung condition. The lung mottling and glandular enlargement have disappeared.

Actinomycosis.—The causative organism is the “ray-fungus” (*Actinomyces bovis*). The type of lesion which it causes in the jaw, mouth, alimentary tract, and elsewhere is well known, and need not be further described. Infection of the lung probably occurs directly through the bronchi. It is usually unilateral.

The four types of *Christison* and *Warwick* are described by Jacobson:

(1) *Bronchitic Type.*—Pus and fungi are present in the bronchi only.

(2) *Pneumonic Type.*—The process spreads to the alveoli, which become filled with pus. This bronchio-pneumonic lesion may become organised, producing a hard fibrous nodule or nodules. In some of the lesions this attempt at repair may fail to occur, and the alveolar walls are destroyed and replaced by pus and fungi.

(3) *Pleuro-pneumonic Type.*—The abscesses grow larger, and the disease spreads to the pleura, where pus or granulation tissue may appear.

(4) *The organism enters the blood-stream and metastasises to other parts of the lung.*

Direct invasive extension is more typical of actinomycosis than of the other mycoses previously described, so that the disease tends to produce one or two extensive lesions rather than multiple scattered foci. A pleuro-pulmonary type is common. The pleura and adjacent lung are matted together by dense fibrotic tissue.

Radiologically, massive consolidations predominate. Abscesses may be demonstrable. Pleural thickening, localised empyema, or extension to

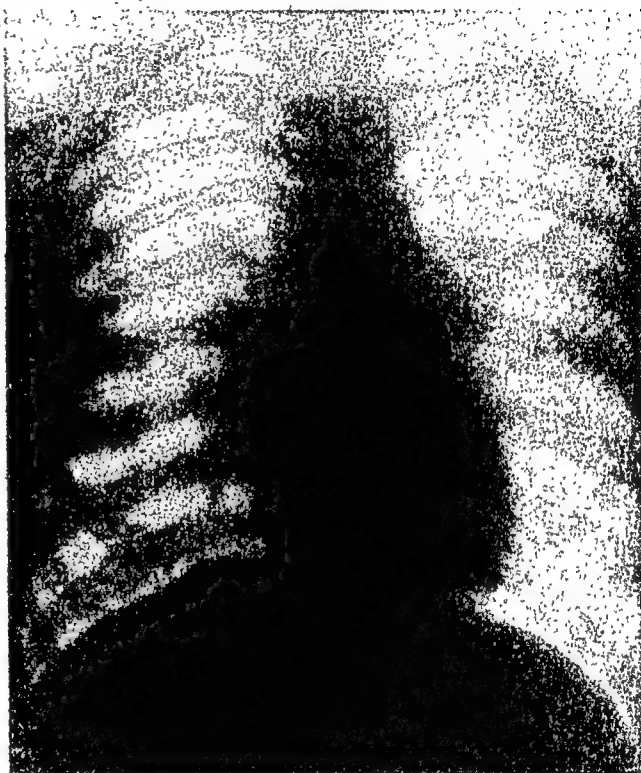


FIG. 181.—Blastomycosis of lung.

thoracic wall may be evident. Discrete nodular involvement of both lungs may occur, though rare. It is usually coarse, but may be miliary in type. A somewhat rare form shows widespread peribronchial thickening with diffuse hazy nodes, giving an impression of interstitial fibrosis. It tends to spread



FIG 182—Actinomycosis of lungs

downwards and to affect the bases more than the apices in contradistinction to tuberculosis.

Streptotrichosis is described by *Pancoast* and *Pendergrass*. It resembles actinomycosis radiologically, but the organism is of branching filamentous type and has different characteristics. It is probably of more frequent incidence than is usually realised. In the lungs it may cause extensive consolidation and multiple cavity formation, pleurisy, and empyema. The lungs are most often affected, but other viscera and the lymph nodes are often attacked.

Torulosis.—The organism is the *Torula histolytica* (*Stoddard* and *Cutler*, 1916). It may produce general or local disease, and primary respiratory

involvement is common. The pulmonary disease is chronic, with a strong tendency to fibrosis, but little to caseation. Pleural involvement is absent or slight. A form with general peribronchial thickening occurs. The lesions are almost always compatible with a diagnosis of tuberculosis.

Aspergillosis.—The species of Aspergillosis associated with pathological conditions in the lungs are *A. fumigatus* and *A. niger*, both commonly found on dead vegetable matter, in various cereals, hay, and straw. Broncho-aspergillosis has been called "bird fanciers' disease," and is prevalent among pigeon and canary fanciers. It has been suggested that infection may occur through the habit of allowing the birds to take food from their lips. It also occurs among hair-sorters. The pathogenicity of this fungus is well established: over 100 cases are on record. The symptoms are those of bronchitis with rather scanty mucopurulent sputum, cyanosis, and marked dyspnoea. Haemoptysis may occur. In some cases the sputum has an odour of brewer's yeast. Radiograms show fine mottling through both lung fields, together with considerable emphysema, superseded later by an accentuation of the lung markings interpreted as fibrosis. The findings are, in fact, those of a bronchitis, and not those of tuberculosis. Gross pneumonic lesions with or without cavitation are also described. Other organs likely to be attacked are the abdominal viscera and mesenteric glands.

Sporotrichosis: infection with sporotrichium, a branching, septate, spore-bearing mycelium. The usual infection is through the skin in agricultural workers and gardeners. After a variable incubation period, a localised skin lesion appears, followed by lymphangitis. The disease may become disseminate in the body, and occasionally attacks the lungs.

Bronchomycosis Feniseiorum.—In 1932 *Munro-Campbell* published a short monograph describing a form of bronchomycosis occurring in Westmorland farm-workers. *Fawcitt*, in the next county, quite independently noted lung changes in a radiogram of the chest of a farm-hand which he traced back to a possible infection from mouldy hay. The man's symptoms dated from a time when he had been moving this hay, from which the dust rose in white clouds. *Fawcitt* later succeeded in identifying five other cases from the radiographic appearances, and submitted the sputa to competent pathologists for examination. The organism found were *Aspergillus fumigatus*, *Penicillium*, and *Mucor*, either alone or mixed. Symptoms came on in some cases in four days after exposure to the dust (corresponding with the time taken for fungus to grow on culture media), in other cases more slowly, when the exposure to the dust had been more prolonged. Dyspnoea was the leading symptom, with some cough and frothy mucopurulent sputum. The clinical signs were those of bronchitis and emphysema. If treated with potassium iodide, the cases improved; the more chronic cases were admitted to sanatoria, but tubercle bacilli were never found. There was only one death: In this fatal case the radiogram, taken three months after the onset of the illness, showed

extensive fine mottling throughout both lungs, with very considerable increase in the hilar and perihilar markings, fading off towards the bases. A film taken five months later showed what appears to be a partial right-sided pneumothorax. There were also small clear areas near the hilum, suggesting cavities or bullæ. The patient did not improve, and three years later died of an acute streptococcal pneumonia. Post-mortem showed emphysema, pleural bullæ, and acute congestive and pneumonic changes from recent infection; there was a considerable amount of fibrosis in bands traversing the lung substance. The exudate contained streptococci and yeast-like cells. The silica content was 1.5 (normal). No tubercle bacilli. The radiological appearances in the other cases were similar. In no case were gross pneumonic or broncho-pneumonic changes visible in the radiograms.

Moniliasis.—The monilias have been described in detail by *Castellani*. Several species are pathogenic to man, and when injected into the lungs of rabbits produce nodular and caseous lesions. *Monilia candida*, a yeast-like organism, has been studied extensively in Jersey by *Marett*, *Stewart*, *Young*, *Wood*, and others. *Marett* believes that infection is carried by milk from infected cows, though this is questioned by other workers. *Oliver* has described the condition as a definite clinical entity. *Monilia* gives rise to pus formation in chronic lung infection. There is a mechanical action due to local growth of the fungi on the mucous membrane, similar to thrush in the mouth. The X-ray findings are as a rule slight, and consist only of increase of the hilar shadows, increased lung markings, and in the form of fans and smudgy streaks running down towards the bases. Bronchiectasis may occur. Treatment is by potassium iodide and vaccines. It is believed by the workers cited that monilia is not only pathogenic by itself, but that its presence in conjunction with tuberculosis seriously prejudices the chances of recovery. *Carler* states that, while moniliasis is generally considered a mild mycosis, the disease varies from a mild bronchitis to a severe disease resembling tuberculosis. One fatal case is recorded.

CHAPTER XXVI

TUBERCULOSIS OF THE LUNGS

GENERAL

THE CONTROVERSY which raged during the earlier years of radiological work over the value of radiology in the study of pulmonary tuberculosis has almost died out, because there is no longer room for doubt that the method properly applied and used will detect the presence of tuberculous disease of the lungs with greater certainty than any other method whatever. Nor is there any question that the negative evidence is reliable, with an extremely high degree of accuracy. Not, be it admitted, with absolute accuracy. Lesions of very small size associated with positive sputum may occasionally be present in situations in which they cannot be detected in a radiogram. Yet even these, in the lifetime of workers of great experience, constitute an almost negligible proportion of the whole.

Assmann records his experience as follows : "Only in extremely rare instances of cases verified by positive sputum tests does the radiological examination show a complete absence of visible change, and in these the density and extent of the lesions may have been too slight to allow of their recognition, or other shadows may have hidden them. The reverse is, however, a matter of daily experience, that tuberculous disease can be demonstrated radiologically when all other clinical means fail." The writer's own experience bears this statement out fully. There have been many occasions on which, owing to some disturbing feature in the history, such as hæmoptysis or pyrexia, he has felt that his expressed opinion that there was no X-ray evidence of disease in the lungs might in the event prove misleading, but he cannot find a single instance in which it has subsequently come to his notice that the patient has developed pulmonary tuberculosis or in which a subsequent radiogram has proved positive.

Samson and Laurason Brown, in an attempt to study the incidence of the five cardinal symptoms in 280 cases of minimal tuberculosis, found in 27 per cent. typical râles, in 27 per cent. hæmoptysis, 12 per cent. effusion, tuberculous bacilli in sputum 35 per cent. Definite parenchymatous lesion was found in 99 per cent. of this group. In a further series of 1,004 consecutive cases, there were none with definite clinical signs and a negative radiogram. There were 396 in which the radiogram showed definite evidence of tuberculosis with normal clinical signs. As regards the extent of the lesion, in 211 cases both methods gave equal evidence. In 361, the radiograms showed more extensive disease than the clinical signs.

But it must be emphasised that conclusions as to the tuberculous aetiology of the disease cannot be drawn from radiological evidence with any complete certainty—since many forms of tuberculosis may occasionally be closely mimicked by other conditions; further, that hard-and-fast opinions upon the activity of the disease are usually unreliable on the evidence of a single or even repeated radiological examination. These points must be determined in the light of all the evidence, and the clinical condition of the patient, the temperature, sputum examination, blood sedimentation, reaction to exercise and fatigue, weight, energy, appetite, and other clinical factors in the picture of the disease must always outweigh the radiological evidence in estimating progress, prognosis, and the need for, and effects of, treatment.

With due regard to these and other limitations of the method, it may be stated, however, that the location and extent, the pattern and the structure, of the tuberculous changes can be determined with great accuracy; that valuable indications as to the age and type of individual lesions may be obtained; that cavitation, fibrosis, extension, or regression of lesions may be determined immediately and unequivocally by this method. The writer has always felt that the controversy referred to at the commencement of this section was singularly inept. He has steadily refused to see any duality of interest in this matter, feeling that it is not of the slightest consequence in any given case which method produces the most abundant or most useful information, provided neither is neglected.

Tuberculosis is often brought to light in patients who are apparently well or have no chest symptoms. It is a common experience of all radiologists to encounter such lesions during the course of a barium-meal examination. *Denker* quotes six cases of tuberculosis discovered among 1,360 healthy policemen, two to six per thousand cases in students, more than half of whom had no suspicions that they were ill. This author believes that a freer use of radiology is an essential part of the fight against tuberculosis. Some physicians have advocated mass examinations of certain classes of the population, namely contact cases, school children and students, inmates of institutions, nurses, policemen, military men, and post-influenzal cases. *Freund* states, from investigation of 20,000 tramway employees, that since 1928 the tuberculous mortality among them has dropped to 6.5 per 10,000, much below the general mortality rate among the better classes. This he ascribes to early X-ray examination available to them since 1928. Prior to that date, the rate among them was the same as that among the general population.

The radiologist who finds, during the course of an opaque-meal examination, evidence of widespread tuberculous involvement has merely obtained easily and with the exercise of quite ordinary care evidence which it was obviously more difficult to obtain clinically. He has no grounds for self-congratulation. The duty of the radiologist, it has always seemed to the writer, is to redouble his efforts and so to improve his knowledge, and to study every particle of evi-

dence which his examination affords, that he shall be in a position to state exactly the meaning and the worth of that evidence. This task is for the writer, as for most of us, as yet unfulfilled. nor will the earnest study of the remaining years of our lives suffice for its fulfilment. The main problem and responsibility remains with the clinician in charge of the case, who is invariably grateful for a reasoned exposition of the radiological findings and usually lenient towards errors which do not proceed from the dogmatism of inexperience on the one hand, or from lack of care on the other.

The need for exact autoptic control of his diagnosis is one which is felt by every radiologist. Unfortunately, very few have the opportunity of seeing the future course of the many tuberculous patients who come within their purview, or of personally comparing radiological with post-mortem findings. An autopsy of a case of pulmonary tuberculosis is, for the generality of practising radiologists, a rare event, unless they work in special chest hospitals or sanatoria, or in public institutions. Radiology owes a debt to the German school of radiologists, notably *Assmann*, *Graeff* and *Küpfertle*. *Simon*, *Redeker*, *Haudek*, *Fleischner*, and others, for a valuable systematisation of knowledge based upon such correlation, which has brought some precision to our conceptions. Though this work still admits of considerable controversy in matters of detail, it has met with sufficient acceptance abroad to warrant an attempt to give an outline of it in this work. Equally valuable work has been done elsewhere on the Continent, and in America the brilliant anatomical researches of *W. S. Miller*, and clinical and radiological studies by *Sante*, *Wessler* and *Jaches*, *Opie*, *Pancoast*, *Pendergrass* and many others, have contributed greatly to our knowledge.

RADIOLOGICAL FINDINGS AND THEIR PATHOLOGICAL BASIS

The Exudative Focal Lesion ("Herd Schatten")

The exudative focus commences as a collection of cells and fluid in one or several acini of the lung. It rapidly extends, and in a very short time produces larger lobular exudative foci. The edge of such a lesion is not sharply defined, since at its periphery air-containing alveoli and alveoli full of exudate may be intermingled, while the surrounding tissue may be the seat of a non-specific perifocal (collateral) reactive process, characterised by hyperæmia and œdema. The absence of any well-defined anatomical boundary is reflected in the X-ray appearances, which show a denser central shadow gradually fading off into its surroundings. Radiologically these foci are rarely single. They usually overlap and are not separated by normally translucent air-containing lung tissue. The picture, therefore, in a case of multiple anatomical lobular exudative lesions, is a rather extensive region of cloudy opacity in which are discovered central, usually somewhat rounded, condensations. The intervening lung, not being completely airless, gives to the whole affected region an

irregular mottled pattern of light and shade. Areas of tuberculous pneumonia involving more extensive areas of lung tissue are not, as a rule, rounded in outline, though they may be; in which case they give rise to great difficulty in diagnosis. Caseation cannot be diagnosed from their extent or density, but only by the occurrence of cavity formation within such areas.

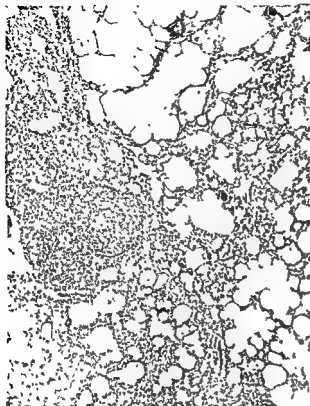


FIG 183.—Section. Tuberculosis of lung Exudative type of lesion $\times 40$

On the left of the section is an organized nodule of tuberculous inflammatory tissue. The alveoli elsewhere are filled with oedematous fluid and cellular exudate, which in a radiogram would cause an ill defined hazy shadow

The shadowed region may not be entirely due to specific tuberculous pneumonia. Collateral inflammatory changes may contribute to the picture; these changes may be due to different factors, such as: (a) infiltration with serum and lymphocytes; (b) fibrinous exudate; (c) desquamation; or (d) epithelioid zones with giant cells. These collateral or "epituberculous" changes have been studied principally during the radiological era. The term "epituberculosis," introduced by *Elias and Neuland* in 1920, has indeed been described as a radiological conception, which in the opinion of some writers has been somewhat over-emphasised; for it has been established that not every fleeting and rapidly clearing perifocal reaction

is an "unspecific" one. True tuberculous pneumonias may behave in the same manner.

Oppenheimer found that a lesion having X-ray appearances like those of epituberculosis followed the experimental introduction of dead tubercle bacilli into the bronchus of rabbits hypersensitive to tuberculosis. In non-allergic animals such results could not be obtained. Intratracheal injections of living bacilli in hypersensitive animals produced lesions radiologically "epituberculous"—anatomically areas of tuberculous pneumonia from which the animals

died. *Oppenheimer* puts forward the theory that the epituberculous lesion is a benign tuberculous pneumonia which resolves ; it is due to a discharge of dead tubercle bacilli and a few living bacilli from a caseous focus ; the progressive lesion, on the other hand, is due to discharge of many living bacilli. The pro-

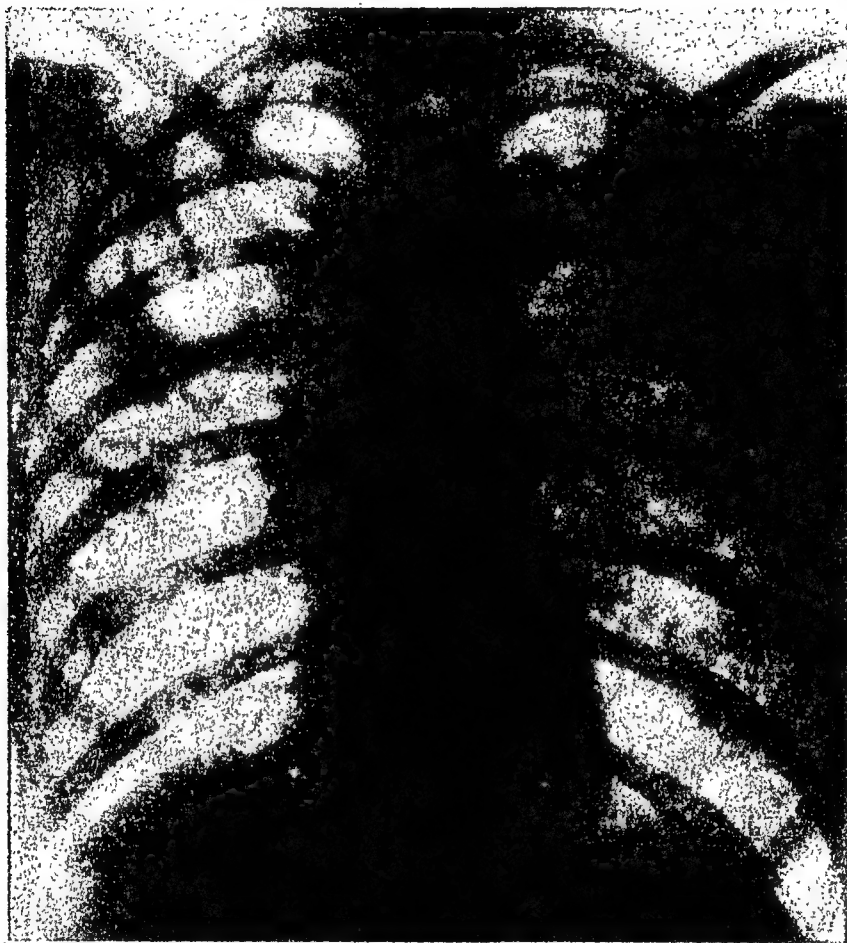


FIG. 184.—Advanced tuberculosis. Mainly exudative with cavitation.
Death one month later.

portion of living to dead bacilli and the degree of resistance of the individual will determine the issue.

Almost any extension of the original focus may show the same type of perifocal reaction, which may clear up rapidly. The existing focus may only be distinguishable by necrosis and cavitation, or later, by calcification.

FURTHER PROGRESS OF EXUDATIVE LESIONS.—(a) If there has been no caseation, they may become absorbed and disappear, leaving no trace visible in the radiogram. (b) If caseation and calcification take place, a dense central

shadow remains. (c) Necrosis and expectoration lead to cavity formation, with a characteristic radiological appearance.

The Productive Focal Lesion

The "productive" focus differs from the exudative in its much more limited size and in its essential pathological nature. It consists of tuberculous granulation tissue, developing in

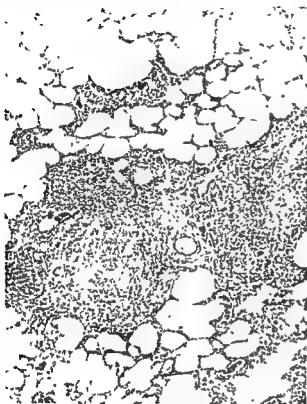
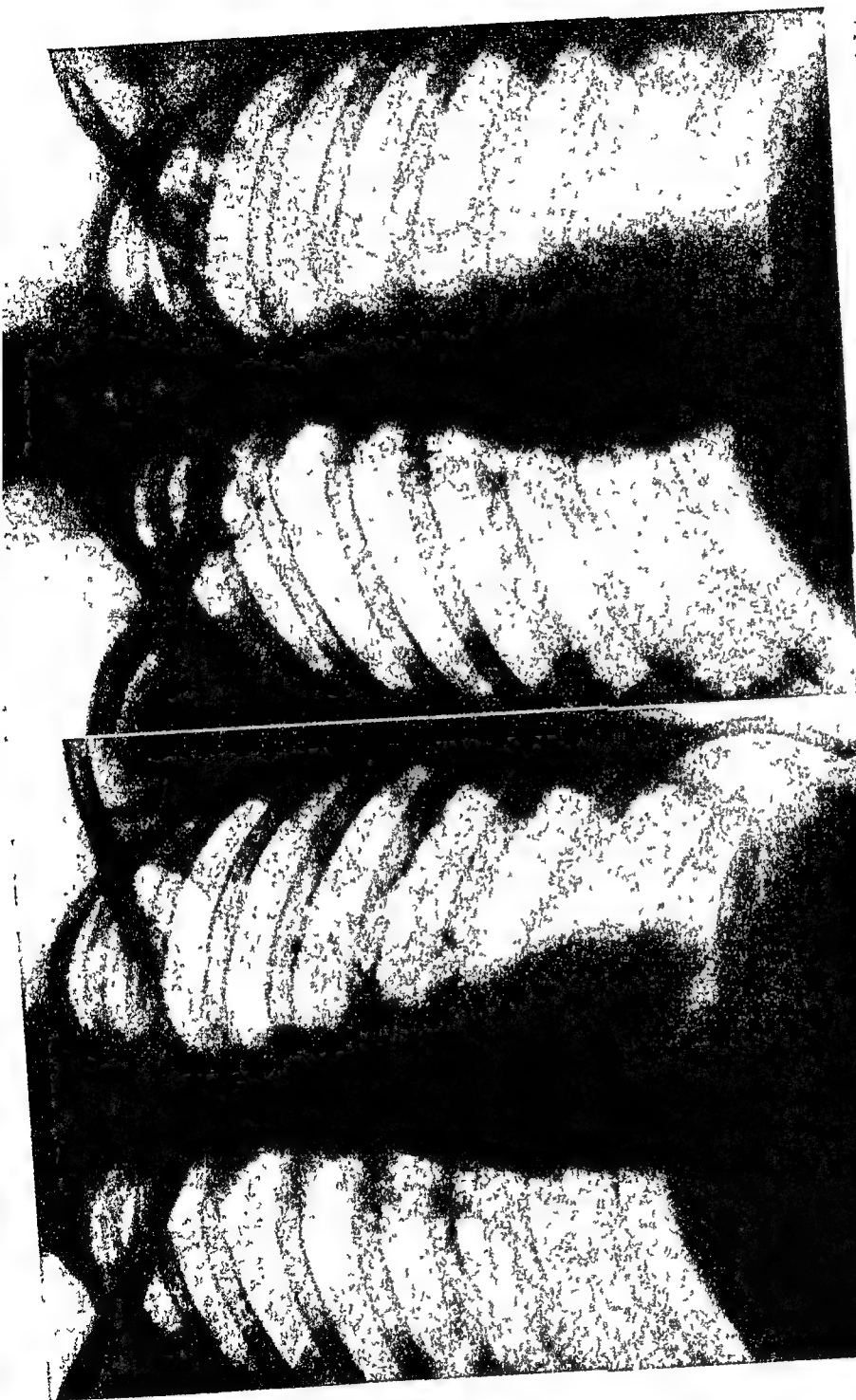


FIG. 185.—Section. Chronic phthisis. Productive type of lesion $\times 40$. The productive lesions consist of organised tuberculous granulation tissue. The lesion is sharply defined from the surrounding alveoli, which contain little exudate. A rosette-like grouping of the lesions may be visible in radiograms. They usually preserve a well defined edge, and there is a marked tendency to fibrosis and calcification.

translucent centre. This ring of opacity can itself be analysed into a group of dots, each about 1 mm. in diameter. This arrangement of the petals is exactly the same, as regards dimensions and general appearance, as the components of the residual shadow found for weeks or months after the administration of

in an acinus of the lung, and invading neighbouring acini. Such foci are not surrounded, like the exudative foci, by zones in which air-containing and exudative-filled alveoli are intermingled. On the contrary, each nodule of tuberculous granulation tissue is surrounded by normal alveoli.

Groups of these foci, often arranged in the form of rosettes, or radiating petals, form a striking picture if careful study is made of a radiogram. They are sharply defined, and surrounded by areas of normally translucent lung. The writer, in an effort to analyse them more closely, has made the following observations. The petals, four to six in number and about $\frac{1}{2}$ inch in diameter, are arranged in rosette form around a central clear dot, a millimetre or two in width. The whole rosette will have a diameter of from $\frac{1}{2}$ to 1 cm. Each petal will be composed of a ring of opacity with



Tuberculosis: same case three years later. Cavity right apex. Productive type of lesions, with fibrosis left apex.

FIG. 186.—Productive tuberculosis both upper lobes.

lipiodol. The lipiodol "rests" in the groups of alveoli in relation to the terminal bronchioles (acini) may show an exact reproduction of the minute structure of productive tuberculosis.

There is no doubt in the writer's mind that the anatomical arrangement is the same in the two cases, and that the same anatomical sub-unit of the lobule is involved in each case (see section "lung parenchyma").

The division into exudative and productive lesions above described represents facts which are certainly true. These different lesions can be recognised radiologically and pathologically. But both forms may be intermixed; and *Assmann* has for this reason cautioned against the too ready application of the terms "productive" and "exudative," stating that in the majority of all cases, productive and exudative processes are going on side by side in the same tuberculous focus, and cannot be separated from one another by the naked eye. If the naked eye cannot distinguish them, he argues that the radiogram cannot.

He prefers the terms "nodular" (*Knötchenförmig*) and "indurative" forms on the one hand, and "caseopneumonic" on the other, which in the main, though not exactly, correspond to the "productive" and "exudative" lesions.

Graeff and *Kupferle* themselves recognise the impossibility of making too sharp a distinction between these two fundamental forms and speak of "mainly productive" or "mainly exudative" lesions. From a prognostic point of view, too much emphasis must not be laid upon the distinction. It is true that many cases with "exudative" characteristics are acute and of a serious type. Also that many, perhaps the majority, with "productive" characteristics are relatively benign, and tend to evolve through fibrosis to healing. Yet we know that many lesions of the exudative type clear up rapidly, and that some of the productive type are serious and progress rapidly. The prognosis too may depend, not on the characteristics of the individual lesions, but upon their extent, and upon the lesions which may develop from them, which can in no way be determined by the character of the lesions present. Other factors of immunity, resistance, and social conditions come into play which, in the present state of knowledge, render nugatory attempts to correlate prognosis with the momentary radiological appearances, except in the most general and tentative way.

Indurative Tuberculosis (Fibrotic)

Both the exudative type and the productive type tend to heal with the formation of a protective fibrous tissue. Evidence of this fibrous reaction is found in the radiograms (1) The fibrosis in and around the tubercles is evidenced by increased density and definition of the individual lesions. (2) The fibrosis in the lung stroma around the lymphatics and blood-vessels, and bronchi, causes a coarse striation of the affected region. The strands run in the direction upwards from the hilum to the periphery. They are coarser and more irregular than the normal vascular shadows, and do not show the same regular

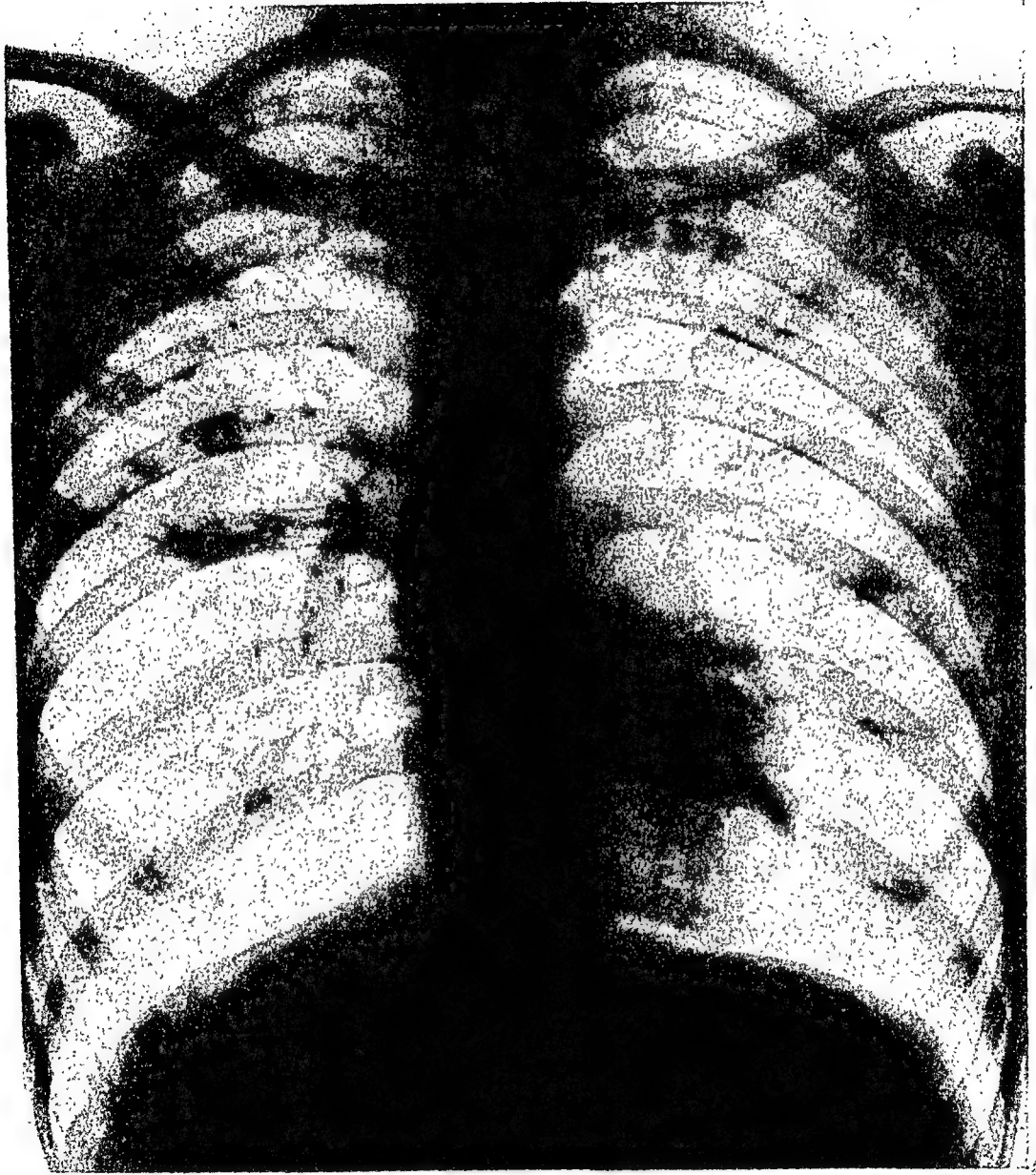


FIG. 187.—Pulmonary tuberculosis—mainly productive. M. 21. Two years' history.
Both parents died of tuberculosis.

branching. The tendency of the fibrous tissue to contract causes these strands to be pulled into straight linear form. The effect upon the surrounding tissues



FIG 188.—Chronic tuberculosis. M 48 Lesions mainly of productive type. Much fibrosis; multiple small cavities, emphysema, tuberculous ulceration of larynx

of the fibrotic contraction is to cause displacement of them in the direction of the zone of induration. Such displacements commonly observed are :

- (1) The trachea is usually pulled towards the lesion, and in advanced apical tuberculosis is convex towards the lesion.
- (2) The affected lobe is diminished in size. The interlobar fissure separating it from other lobes is displaced towards the lesion, and usually concave.
- (3) If the lesion is in the upper lobe, there is frequently a displacement of the hilum upwards on the affected side. The vascular trunks then originate at

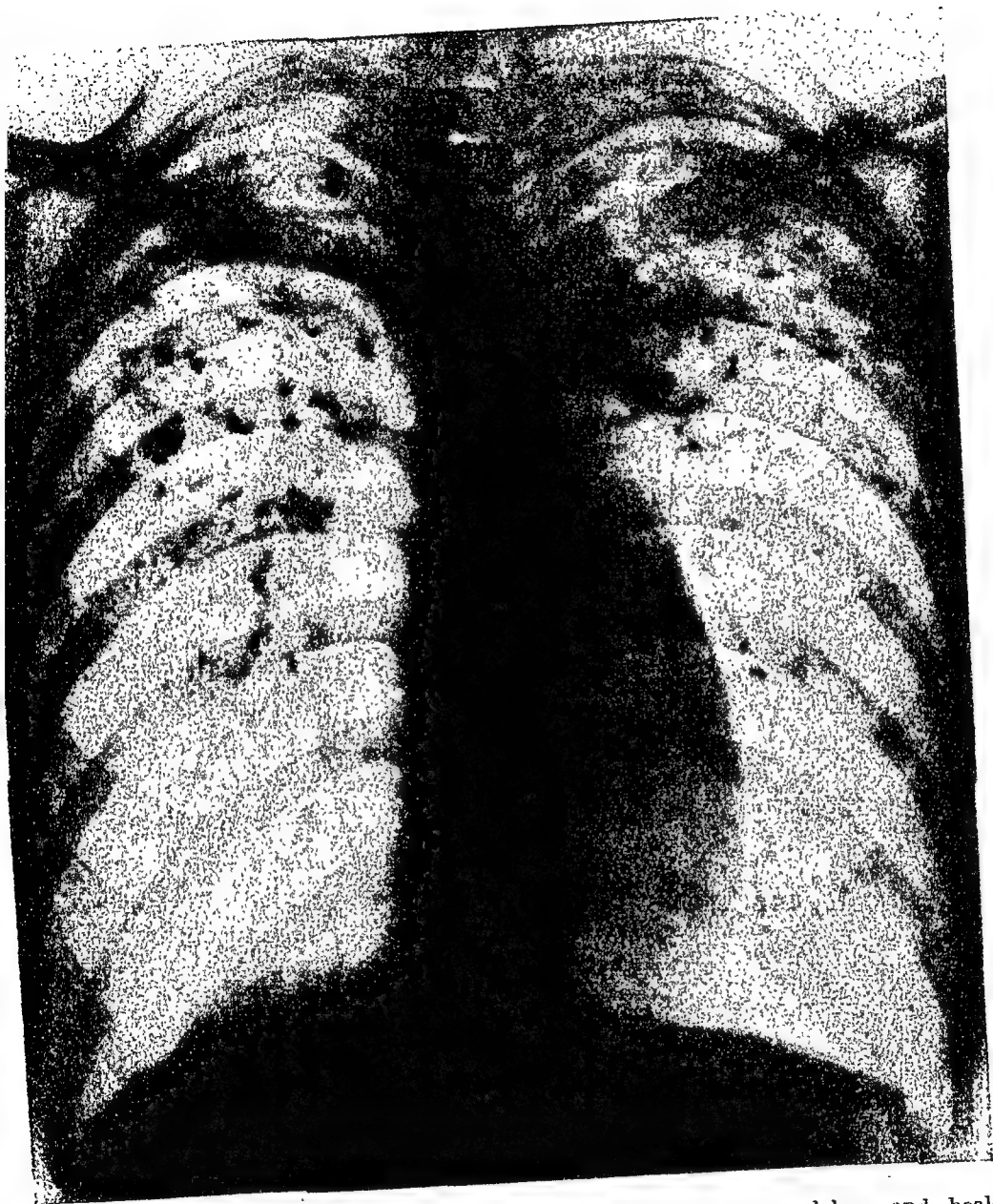


FIG. 189.—Old tuberculosis. Calcified healed lesions in both upper lobes, and healed tuberculous bronchopneumonic foci left base.

a point higher than normal and are directed almost vertically downwards towards the base. A dissociation of the components of the hilar shadow is often seen, and the pulmonary arteries and their branches stand out with unusual distinctness.

If the fibrosis is of the massive type, the affected lobe may be shrunken and



FIG. 100.—Pulmonary tuberculosis: Fibrosis and cavitation right upper lobe. Disseminate bronchopneumonic lesions in lower lobes.

airless, either throughout its extent or through a wedge-shaped area which, in the lateral view, may correspond with the distribution of a principal subdivision of the lobar bronchus. In extensive fibrosis, the chest wall falls in, and the ribs slope more steeply on the affected side. The diaphragm is elevated and the heart and mediastinum displaced towards the affected side. A complete

unilateral obscurity, with this displacement of the diaphragm and mediastinal structures, often combined with thickening of the pleura, is sometimes encountered as an end result of tuberculosis. It may be indistinguishable from the very similar picture due to bronchial carcinoma with unilateral complete atelectasis, or from the end result of a chronic interstitial pneumonia.

Emphysema is usually present, (a) as a compensatory emphysema of the unaffected lobes; (b) in the alveoli of the diseased area which have not been affected by the disease. This emphysema is usually a marked feature, and produces a clearly defined mottling of translucent areas intermingled with, or bordering upon, the densities due to the disease. When these emphysematous areas are large and of rounded form, they simulate cavities: indeed, it is often impossible to distinguish one from the other without lipiodol.

Cavitation

Cavitation occurs as a result of the central necrosis of a caseous area, which may be actually very small, or large. A small cavity may rapidly increase in size as a result of elastic traction of the surrounding lung; this mechanism is proved by the diminution in its size which follows artificial pneumothorax, and has been convincingly demonstrated by *Fleischner* in radiograms showing marked variation in size of a thin-walled cavity in inspiration and expiration. Another mode of formation is the rapid breaking down of a large area of caseation and expectoration of the contents.

Morland, however, believes that the thin-walled ring shadows are more often caused by air under pressure following partial obstruction of a small bronchial tube which would link them with the large annular shadows due to emphysematous bullæ. He quotes *Vere Pearson*, who found positive pressure after introduction of a pneumothorax needle into a large thick-walled cavity, which, after reduction to atmospheric pressure, again showed a positive pressure when the patient breathed deeply, proving the presence of a valvular opening. Positive pressure has also been found in large emphysematous bullæ (*Hochsinger*). *Morland* thinks that a small layer of fluid covering the orifice of a bronchus leading to a cavity might cause a valvular obstruction by allowing air to bubble through it into a cavity while preventing its exit because the secretion is too tenacious to escape through the narrow opening. The two theories are not irreconcilable, but it would certainly appear that the elastic traction of the lung during inspiration is in a direction away from the centre of the cavity and that, even during expiration, the elastic force acts in the same direction, though with diminished power, tending to keep the cavity open and not to close it. Yet it is a matter of common observation that acute cavities, whether tuberculous or due to abscess, do close and rapidly disappear, in spite of this elastic traction. The explanation can be found, perhaps, in the rapid development of compensatory emphysema of other portions of the lung of a degree sufficient to counteract this force.

RADIOLOGICAL APPEARANCES.—When the necrotic material in the centre of the tuberculous area has been replaced by air, but not before, it becomes visible in a radiogram as a circular, oval, or irregularly crenated shadow of varying extent. It is extremely important to verify the fact of cavity formation, since it is of serious prognostic significance.

Several different types may be observed :

(1) *Early Cavity.*—A cavity is not necessarily evidence of chronicity. Some early cases of tuberculosis present themselves with a single apical cavity.



FIG. 191.—Pulmonary tuberculosis Apical cavity.

as the sole radiological manifestation. The cavity resulting from the rapid breakdown and complete evacuation of an early infiltration will show as a circular translucency with a delicate wall ; there may be slight thickening of the trunk which supplies it, due to lymphangitis of the vessels leading from it to the hilum. A fluid-level may be present, but is more likely to be absent. Such an early cavity may be in the upper lobe (usually posteriorly) or in the apex of the lower lobe. These are sites of election. Such early cavities may diminish in size rapidly and disappear in a few weeks or months, or may be followed by rapid bronchogenic dissemination.

(2) *Cavities of recent origin*, often multiple, may occur in the centre of a region of tuberculous infiltration which is cascating. These cavities are diffi-

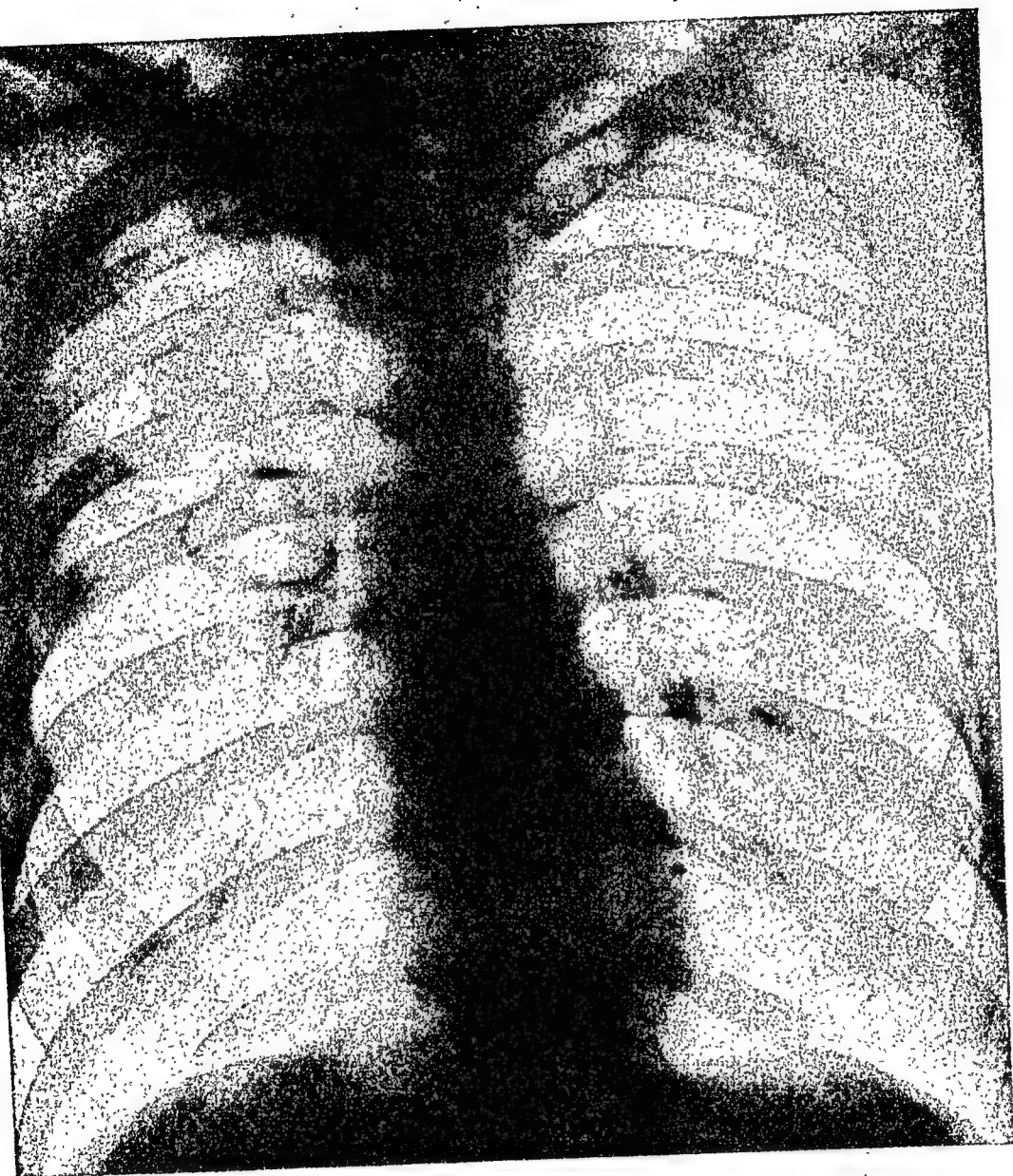


FIG. 192.—Pulmonary tuberculosis with cavitation. The “hilar” cavity was in the apex of the right lower lobe.

cult to detect because they are obscured by the surrounding shadow, and their walls, being formed of a considerable extent of infiltrated lung, are only defined on their central side by the faint air shadow, and are ill-defined peripherally.

(3) *Ring shadows*, having a clearly defined wall, which may vary from one or two millimetres to a quarter of an inch in thickness. These cavities may be multiple. They are usually chronic, but not invariably. The wall may be

fibrous, or composed of softened pulmonary tissue.

(4) *Small cavities in a mass of fibroid or fibrocaseous lung tissue* present a difficult problem, since they are masked or simulated by emphysematous areas in the same region. It is probable that tomography will be of great value in such cases.

FLUID-LEVELS IN TUBERCULOUS CAVITIES.—Owing to their unusual position in the upper parts of the lung, the cavities drain relatively well; incidental blocking of the draining bronchus seems to occur but rarely; so that fluid-levels may be absent or inconspicuous at the time of examination. A very careful inspection of the lower contour of the cavity will, however, often detect a slight flatten-

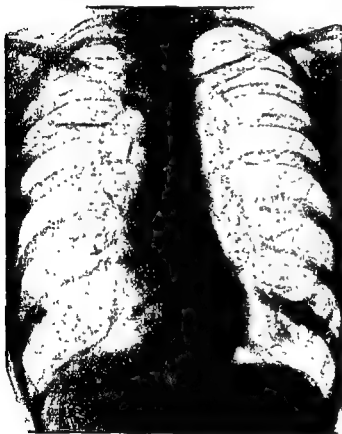


FIG 193.—Tuberculosis with cavity right apex. Note shallow layer of fluid in cavity. F 34. Symptoms, paroxysmal attacks of coughing following influenza 1½ years previously. Sputum positive.

ing of this contour, due to a small layer of fluid, and this meniscus will remain horizontal while the patient is inclined to one side.

DIFFERENTIAL DIAGNOSIS OF CAVITATION.—The following conditions should be borne in mind:

(1) Emphysema in a fibrotic tuberculous area.

(2) Emphysematous bullæ. It has been demonstrated that bullous subpleural emphysema will give rise to cavity-like shadows. A rather similar appearance of lobulated thin-walled cavities occurs in one form of congenital cystic disease of the lung; this condition is distinguishable by the thinness and delicacy of the septa.

(3) There are numerous conditions giving rise to cavity formation which may on occasion need to be differentiated from tuberculous disease. Mention may be made of (1) abscess; (2) cavernous types of pulmonary neoplasm; (3) ruptured hydatid cyst; (4) solitary congenital lung cyst.

Cavities may sometimes be simulated, particularly in the right hilar region, by trunk shadows which have roughly circular distribution. Re-examination with the patient turned to a different angle will usually make the distinction clear.

LOCALISED PNEUMOTHORAX.—The suggestion entertained formerly that many so-called cavities were localised pneumothoraces has almost received its quietus as a result of the extended use of therapeutic pneumothorax, which in nearly every instance shows that the ring shadow is situated within the collapsed lung. Cases of difficulty do, however, arise at the extreme apex. It is possible for a localised pneumothorax to form at the apex which can be distinguished only with difficulty or not at all from an extensive cavity. In one case observed by the writer, of bilateral spontaneous apical pneumothorax, the diagnosis rested upon (a) the absence of any apical cap of thickening over the translucent area; (b) its shape, which was not rounded, but bounded below by flattened lung; (c) the presence in the lung below the pneumothorax of the trunks of supply of the whole apex, condensed and flattened by the presence of the air in the pneumothorax cavity.

DIFFERENTIAL DIAGNOSIS OF TUBERCULOSIS OF THE LUNGS

STREPTOCOCCAL OR INFLUENZAL PULMONARY INFECTIONS may simulate tuberculosis. These may be acute or chronic, and the differentiation from phthisis is made, as a rule, by the site of the lesion. The basal lesion with free apex should be considered non-tuberculous unless the sputum is positive for tubercle bacilli, or the X-ray picture shows something characteristic of tuberculosis. Basal tuberculosis is comparatively rare.

BRONCHIECTASIS may clinically be easily mistaken for tuberculosis, particularly if there is hæmoptysis, which is often present in the bronchiectatic patient. But in phthisical patients tubercle bacilli and elastic fibres are almost always found when the sputum is profuse, and patients with large tuberculous cavities are usually febrile and cachectic. Radiologically the localisation of a tuberculous lesion in the upper lobe and the bronchiectatic in the lower or middle lobe is a most important distinguishing feature. The distinction can usually be made by lipiodol.

LUNG ABSCESS.—This can usually be differentiated from tuberculosis by consideration of the history, abscess beginning with an attack of aspiration pneumonia very frequently after operation on the mouth or following some

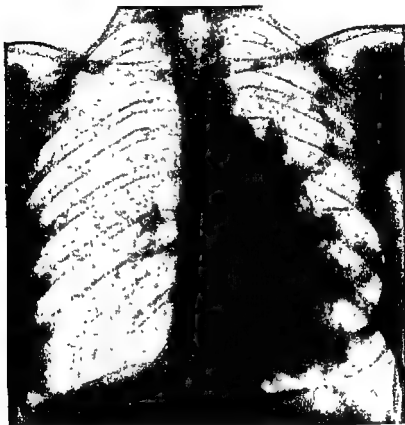


FIG 191.—Pulmonary tuberculosis with cavitation left upper lobe. F 40 Admitted to sanatorium four months previously Sputum positive

septic disease, e.g. appendicitis. The sputum is usually brownish and foul. Haemoptysis may occur.

NEOPLASM. The age of incidence of cancer of the lung, which is very rare under 35, is the principal distinguishing feature. In carcinoma of the bronchus the first symptoms usually occur after 40. Cough, pain, dyspnoea, and signs of cachexia are the principal clinical signs. The radiological findings are as a rule distinctive, collapse of a large portion of the lung with hilar enlargement and sometimes pleural effusion. There are, however, occasionally found cases of senile tuberculosis, particularly of the right upper lobe, with fibrotic collapse of the lobe and homogeneous shadowing, which are difficult to differentiate.

In carcinoma, the greatest opacity is usually in the region nearest the hilum, being caused by the growth and the atelectasis, while the peripheral region of the lung is less opaque (atelectasis or partial atelectasis only). In tuberculosis the principal opacity is more often peripheral, and the peripheral opacity is less homogeneous.

ACTINOMYCOSIS.—This is usually in the lower or middle lobe, but if occurring in the upper lobe may be quite indistinguishable from tuberculosis, unless ray fungus is present in the sputum.

INFECTIONS WITH STREPTOTHRIX and by **BRONCHO-SPIROCHÆTOSIS** produce symptoms similar to tuberculosis, and can only be distinguished bacteriologically.

SYPHILIS of the lung is rare, and is usually in the lower or middle lobe, apex remaining practically free. The Wassermann reaction may help, but is frequently positive in tuberculosis. Moreover, the two diseases may be concurrent. The therapeutic test by administration of anti-syphilitic remedies may be the only distinguishing feature.

Differential Diagnosis of the Assmann Focus

The sharply defined Assmann focus (see p. 299) may closely resemble (1) a primary or metastatic tumour; (2) abscess; (3) infarct; (4) syphilis (gumma); (5) actinomycosis; (6) cyst.

In rare instances, localised rounded bronchopneumonic patches occur in similar situations in influenza, but they are usually less sharply contoured, and clear up more rapidly. Assmann emphasises that the tuberculous ætiology cannot be safely assumed in the presence of an "Assmann" focus.

Kerley draws attention to rounded shadows which may be apical, closely simulating an Assmann's focus in thrombo-phlebitis migrans. The patient, suffering from phlebitis of veins of the legs, develops pain in the chest and slight dyspnoea, with sudden pyrexia. The radiogram reveals the appearances already mentioned, which usually clear up rapidly in a few weeks. Their rapidity of evolution tends to distinguish them from tuberculous lesions.

A rounded shadow, non-pulsating, due to dilatation of a pulmonary vein, is described by *Jacchia*; another due to aneurysm of the pulmonary artery by *Lüdin*. Multiple pulmonary aneurysms may occur.

Seth Hirsch found multiple round shadows in polycythæmia vera. These are often transient and may disappear in three weeks. The pathology is obscure, but he believes them to be due to subpleural thrombi, or small hæmorrhages.

It must be emphasised that the Assmann's round focus is a comparatively rare finding—and that the more chronic rounded tuberculous foci are also rare.

CHAPTER XXVII

TUBERCULOSIS IN ADULTS

GENERAL

THE ONSET of tuberculosis is relatively infrequent in adults after the age of 25. *Habbe, Brown, and Sampson* believe that chronic pulmonary tuberculosis very rarely develops in individuals who had radiologically normal lungs at the age of about 25. From puberty to 25 is the age of most frequent incidence; children occasionally show lesions of the adult type. The trend of modern thought is towards the acceptance of the view that tuberculosis in the adult is usually a reinfection of an individual who has been previously infected in childhood and many believe that such childhood infection is almost universal under civilised conditions. There is a good deal of evidence that adult reinfection is also extremely common, since in the great majority of adults (over 90 per cent according to *Aschoff*) there is present, in addition to the primary focus, another lesion in the lungs, which may later take the form of a subpleural scar, hard and often calcifying, or a calcareous lesion embedded in the lung (*Puhl, Focke, Loeschle*). It is disputed whether the onset of "adult" tuberculosis is the result of a new exogenous infection or an endogenous infection from bacilli shut up in a focus acquired in childhood. It seems certain that the manifestations of the disease in adult type have been modified profoundly by a previous childhood infection. As regards the difference between adult and childhood tuberculosis, certain points stand out clearly:

- (1) The apical (upper lobe or apex of lower lobe) involvement in adults contrasted with children, in whom the first lung lesions may be anywhere, and is often basal.
- (2) The non-involvement of bronchial and mediastinal glands in the adult contrasted with its great frequency in childhood
- (3) The relatively chronic course in adults as compared with children.
- (4) The relatively "typical" radiological appearances in adults, in whom the diagnosis is usually easy. In children the disease may be quite indistinguishable from infective lesions of other aetiology.

EARLY APICAL TUBERCULOSIS

The first lesions of adult tuberculous infection are found most frequently in the subclavicular region. The lesion is not, as a rule, on the anterior aspect, but in the dorsal subapical region of the upper lobe (*Assmann*), and its deep situation may be proved by suitable means. The writer has devised for this

purpose a modified lateral view of the apices described earlier in this section. This infraclavicular region must be scrutinised with particular care, since the shadows of an early lesion may be delicate and limited in extent. It is not the exclusive site of origin. Other less frequent sites are indicated diagrammatically below (*Haudek*).

In two cases the writer has seen isolated tuberculous lesions at the base situated in the lower lobe and spreading along the main fissure. It is important to note that in one of these the lesion showed no evidence whatever in the plain postero-anterior film, but was readily shown in the hollow-back film and just visible in the lateral.

ASSOCIATION WITH DIABETES.—A florid pneumonic type of tuberculosis occurs in diabetics. The middle regions of the lung tend to be affected chiefly, and the “scissural” type, extending outward in the base of the right upper lobe, appears to be common. Cavitation is frequent.

Radiological Appearances.—Various types occur :

(1) **THE FAN-SHAPED LESION.**—A delicate shadow of triangular shape with its base at the periphery and its apex directed towards the hilum is a common

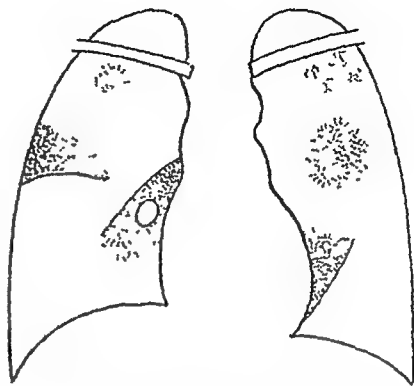


FIG. 195.—Diagram : Some common sites of origin of adult tuberculosis : infraclavicular, base of right upper lobe, apices of lower lobes, and lingual tip of left upper lobe (relatively rare).



FIG. 196.—Early tuberculosis, left apex.

finding. The shadow has been called “Dunham’s fan.” The fan shape is not distinctive of tuberculosis, but is a common feature of many diseases affecting an area of parenchyma through the bronchial or vascular supply. The lesion is cone-shaped rather than fan-shaped ; this shape is inherent in the arboreal

structure of the lung and is a familiar sight in nature in trees and plants, whenever a bough, branch, or twig is damaged or permeated by disease. The triangle is an axial section of the cone. Should the axis of the cone be directed in the line of the rays, the shadow will be approximately circular. If it has an



FIG 197—Early pulmonary tuberculosis, left apex.

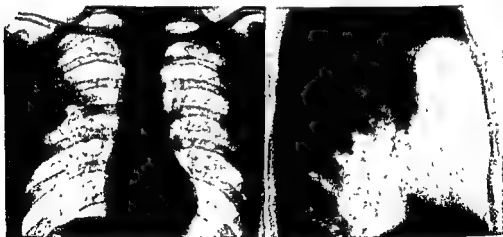


FIG 198—Tuberculosis right upper lobe
M 23

Lateral view. Posterior apical segment involved. Note limitation by oblique interlobe.

intermediate direction the shadow will approximate in shape to other sections of the cone (ellipse, parabola). The structure of the shadow varies. It may be homogeneous, if the lesion is seen in a stage of "infiltration," or broken up into delicate nodules with intervening strands if productive changes predom-

inate. The former is the rule in very early lesions. Clinically fever and malaise are often present, and the symptoms of onset may frequently be those of an influenza or feverish cold. In other cases the onset may be insidious. The early lesion may develop in a few days to a quite extensive patch of shadow.

(2) **ROUNDED FOCUS.**—About twelve years ago, *Assmann* found, in a series of doctors and nurses, early lesions in the lateral infraclavicular region,

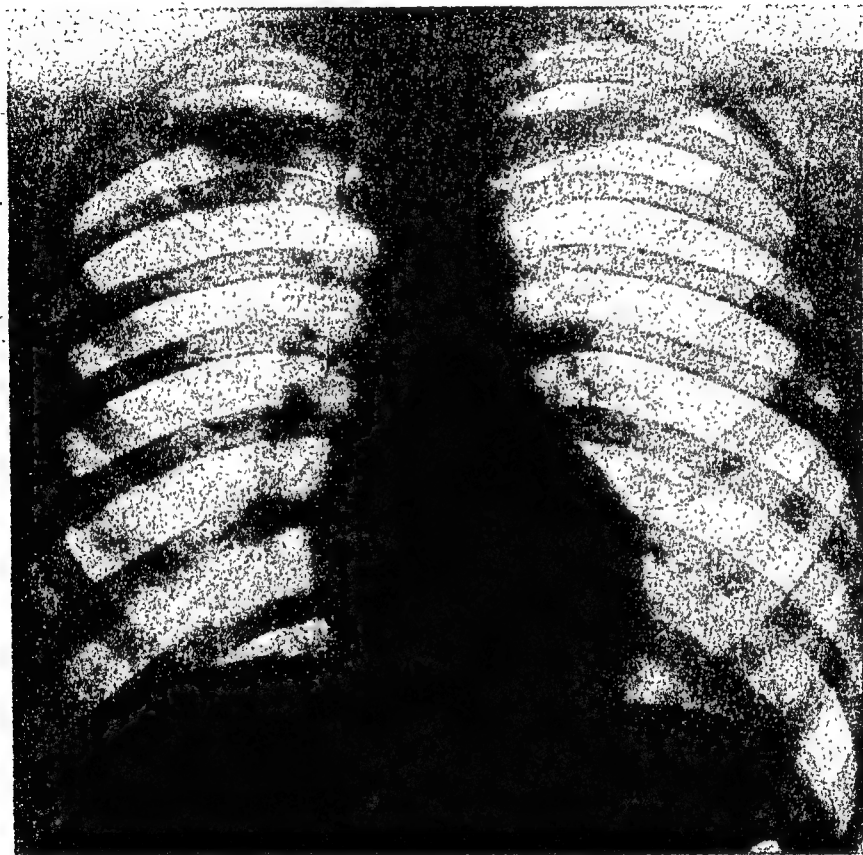


FIG. 199.—Pulmonary tuberculosis. Assmann's focus in right infraclavicular region. Sputum positive.

representing the acute or subacute initial stage of tuberculous infection. Most of the patients were in the third decade of life. The radiogram showed round homogeneous shadows in the dorsal subapical region, varying in size from a halfpenny to half a crown, which were sometimes blurred at the edges, sometimes sharply defined. In other cases there was a more or less diffuse loss of translucency gradually fading off into the lung field (perifocal infiltration). Other sites are the apex of the lower lobe or the paravertebral region. The anatomical basis is a localised tuberculous pneumonia (exudative,

lesion) with a marked tendency to central caseation, which may lead to the rapid production of an early cavity.

(3) **DISCRETE MOTTINGS.**—*Wessler* and *Jaches* described groups of small round mottlings between the clavicles in early cases. This is a type which is not uncommonly observed. It should be emphasised that these first infections are not often seen in their earliest stages, nor is it usual to find very small lesions or first infections. Most of the cases present, at first examination, evidence of rather extensive involvement. It is unusual to find the involved area smaller than a florin, and, below this size, the smaller the lesion, the less likely is it to be "early."

THE SUPRACLAVICULAR APICAL LESION.—A first infection may occur above the clavicle, but this is rare. Most of the supraclavicular apical lesions are older, and many are residual. Residual changes at the apex take the following forms:

(1) Thickening of the pleura over the apex (pleural cap—*Van Zwaluwenburg*).

(2) Crenation of the apical border of the lung; the summit of the lung is puckered, and fibrous strands dip downwards into the lung, sometimes terminating in a faint "flare" of shadow which is usually due to pleural and subpleural thickening. These appearances nearly always go hand in hand with retraction of the apex.

(3) *Simon's foci*. Small rounded or irregular, sharply defined, and sometimes calcified spots in the supraclavicular apex. Some of these result from rests of a previous puerile attack with hæmatogenous spread; others represent healed infraclavicular lesions which have been pulled upwards into the supraclavicular field by fibrosis at the apex. These lesions are almost invariably of old tuberculous origin, and if carefully observed over a considerable time rarely alter or become reactivated. Fresh lesions may, however, develop elsewhere either in the same lung or at a distance.

Further Progress of "Early" Lesion: Adult Type

The lesion may, in rare instances, clear up rapidly and in a short time fail to show any trace of its presence. In other cases there is a healing by scar tissue, and calcification of the central focus. The scarring leads to a dense fibrotic focus, or the disease may extend locally. More often the disease progresses with cavity formation and the production of new foci in the neighbourhood or elsewhere by aspiration. The secondary infiltration elsewhere in the lung tends to affect the lung adjoining interlobar fissures (scissural form). Triangular, cone-shaped, or pyramidal lesions occur with considerable frequency. Each fresh implantation may be accompanied, as *Haudek* has pointed out, by fresh symptoms. The spread of tuberculosis, as studied radiographically, contradicts the old ideas of a steady insidious spread from apex downwards. Rather does it appear to extend by a series of catastrophes



FIG. 200A.—Early tuberculosis. F. 20. Sputum negative, but clinical signs suspicious. Radiogram shows an exudative lesion in the left infraclavicular region.

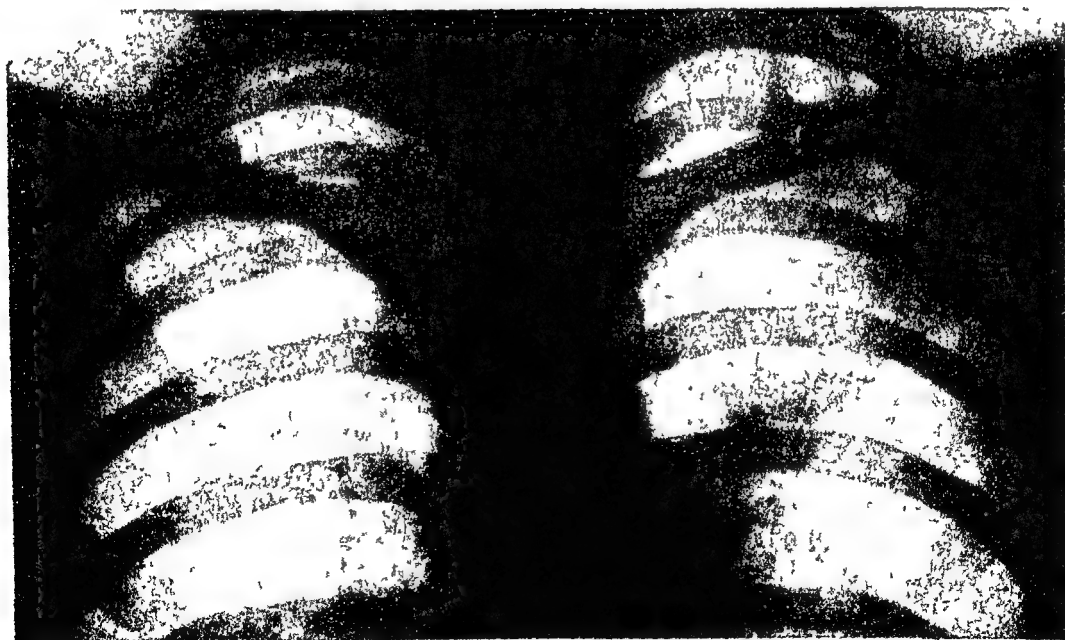


FIG. 200B.—Same case nine months later. Extension of the diseased area. A shadow composed of numerous fine strands now connects the infraclavicular focus with the hilum. (Peribronchial tuberculous lymphangitis.) Sputum positive.

It is a striking fact, if cases of long standing and chronic tuberculosis are studied, how frequently the picture is one of multiplicity of lesions, manifestly of different ages—some calcified, others in various stages of fibrosis, some recent

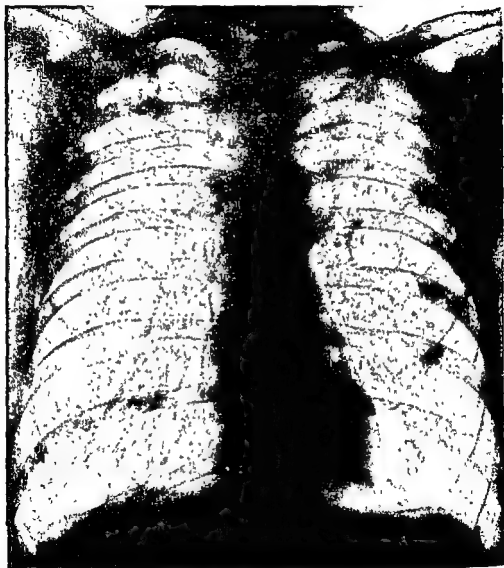


FIG. 201—Pulmonary tuberculosis

—in the upper lobes; frequently there are showers of bronchopneumonic lesions at the bases, which may in turn show evidence of healing, fibrosis, or calcification.

In unfavourable cases the lesions may extend rapidly, producing a large area of pneumonic infiltration which is only distinguishable from a pneumonic shadow by its more mottled density.

Differential diagnosis of tuberculosis of the lungs from pneumonia of influenzal origin is sometimes impossible without repeated observation.

- EXTENSIVE TUBERCULOSIS

Mainly Productive Type (Nodular Form)

The great majority of patients giving positive radiological findings of tuberculosis of the lungs have lesions of considerable extent. This applies also to cases which are clinically early, or merely "suspicious," and to many patients in whom the discovery of the disease is accidental. Of these latent cases the majority show lesions mainly of the productive type: that is, in the form of small nodulations separated by areas of translucent lung tissue, often with strands of fibrosis leading from the lesion to the hilum and also permeating the lesion. It is this fact which gives rise to the impression, in part justified, that this type of lesion is more benign. It is, however, true that some patients with "productive" lesions are seriously ill, and no very definite deductions can be drawn from the radiological appearances. In general this type runs a chronic course. Cavities may be present in the diseased area. Their appearance has already been discussed.

Mainly Exudative Type

A predominance of woolly, ill-defined, homogeneous dense areas of shadow denotes the presence of exudative lobular pneumonia, a special form of bronchopneumonic inflammatory process in which many adjacent lobules are involved at the same time. The foci are multiple, and the shadows coalesce, sometimes by overlapping, sometimes by direct continuity. The tendency to cavitation in these areas, and the relative absence of protective fibrous tissue reaction around the foci, as well as the fact that large areas of lung are usually affected, give to this type of disease a serious prognostic significance.

COURSE.—The lesions may coalesce, and involve the greater part of a lobe. Caseation and cavitation are common. The lesions are not, however, always caseous, but may be of a gelatinous nature capable of complete absorption.

Lobar Type

Essentially this is the same as that first described, but involves a whole lobe, or the greater part of it. The right upper lobe is most often involved. The alveoli are filled with exudate, and little air enters the lobe. There is frequently evidence of partial atelectasis, with displacement of the interlobar fissure towards the affected lobe.

Barjon long ago pointed out the frequency with which this type is found in the neighbourhood of the interlobar fissures, and this has been commented upon

by other writers (*Fleischner, Haudek*). A favourite site is the interlobar fissure between upper and middle lobes, the disease occupying the lower part of the upper lobe, anteriorly. Involvement of the posterior part of the right upper lobe, limited below by the upper part of the main interlobar fissure, may

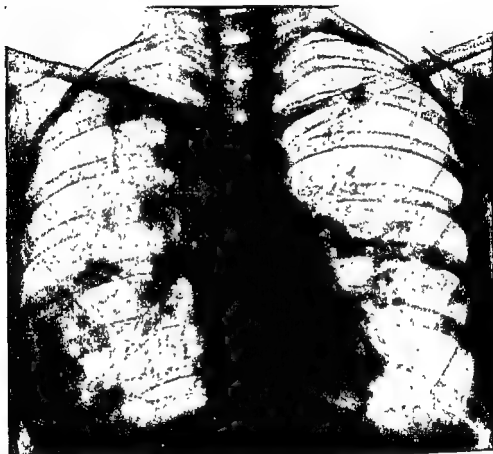


FIG. 202.—Caverno casous tuberculosis. Typical exudative lesions. F. 19. Seven years diabetes melitus. Recent symptoms of active phthisis.

also occur, or in other cases the lateral view may show both regions to be infiltrated by the disease.

Atelectasis

Westermarck, in a paper read before the IVth International Congress of Radiology in Zürich, 1934, showed that atelectasis is a common complication of tuberculosis of the lungs, and if this element is looked for in the radiogram, it will frequently be found.

atelectasis is due to bronchial obstruction and may occur—
(b) Through blocking of the lumen by blood-clot as a result of hæmoptysis (commonest cause). In this case the blood-clot may be coughed up or



FIG. 203.—Pulmonary tuberculosis. Simulating diffuse type of secondary malignancy.

absorbed, and the atelectasis is transient. Mucoid or purulent secretion may act in a similar way, and the atelectasis from this cause is also temporary.
(b) Changes in the bronchial wall. *Westermarck* has carried out a series of investigations in this most important group. Post-mortem bronchial in-

jections with lipiodol and radiograms compared carefully with the dissections in some twenty necropsies on tuberculous patients showed that the bronchi were occluded in the diseased areas of lung, except where they communicated with cavities. The occlusion was due to pus, caseous necrotic granulation tissue, or both. Granulation tissue predominated in the more chronic cases



FIG. 204.—Tuberculosis, right apex Caseous with cavitation Acute tuberculous bronchopneumonia at left base.

The process is a tuberculous ulcerative bronchitis. When the affected bronchi were opened, their branches were found to be similarly occluded.

(c) A third cause of atelectasis is pressure on the bronchi by enlarged hilar glands. This is usually of wide extent, but may involve only one lobe or a part of a lobe. It is commonest in children.

The atelectases have certain common sites—namely in the anterior and

posterior basal regions of the right upper lobe. In the lateral view these show as wedges of shadow; with their apices at the hilum and their bases directed to the periphery. Their appearance is striking. The interlobar fissure is curved towards the collapsed region.

Westermarck has shown that if an effusion exists, the presence of the atelectasis may affect the shape of the shadow of the effusion; the upper border is higher on the medial than the lateral side, so that the slope of the upper border of the effusion is downwards and outwards, reversing the usual appearance.

Fresh exudative tuberculous changes are often associated with large or small patches of atelectasis. Chronic fibrous tuberculosis, with contraction of the fibrous tissue and pleural thickening, may give similar appearances, but the shadow is less homogeneous and less dense. It is unknown whether the effect of this form of atelectasis is favourable to the patient or not. *Striesselmann* suggests that it acts protectively, while *Westermarck* considers it may favour the formation of bronchiectasis and cavitation, and therefore should be treated by artificial pneumothorax.

It would appear to the writer that the combined result of the atelectasis and bronchial obstruction, by preventing the ebb and flow of air through the diseased atelectatic area in breathing and coughing, and by limiting the output of purulent secretion, would on the whole tend to limit the spread of infection through the bronchi to other regions of the lung: and, moreover, that it would favour healing by setting a diseased portion of the lung at rest. According to *Naveau* and *Pesque*, if it occurs in a lobe which contains cavities, it favours cicatrization and cure. If it occurs in another lobe, it increases the negative pleural pressure and may aggravate the disease in the affected lobe.

This is borne out by the experimental work of *Adams* and *Vorwald*. After occlusion of a bronchus by cauterisation, they injected active tubercle bacilli; fewer lesions developed in the collapsed lobe than elsewhere in the lung, and these lesions failed to progress and often healed in the collapsed lobe, though active in the aerated portion of the lung. They concluded that atelectasis of pulmonary tissue exercised a favourable effect on experimental pulmonary tuberculosis in dogs.

Sokol and *Eloesser* have also drawn attention to massive atelectasis occurring as an acute or subacute febrile complication of tuberculosis.

Fibroid (Indurative) Tuberculosis

Healing of tuberculous lesions takes place by the formation of fibrous tissue; fibrosis is never an initial lesion, but must always follow or coexist with the other forms, and is especially frequent in the "productive type," in which granulation tissue is formed at the outset. The fibrosis tends to surround and encapsule the healing lesion, and to prevent direct extension to neighbouring lung tissue. It further rests the diseased lung by diminishing its volume.

Radiological Appearances

Individual foci when fibrotic, become smaller and more sharply defined. If extensive, the lesion is broken up by strands which have a radiating pattern and tend to converge towards the hilum, to which they are often fixed by strands of peribronchitic thickening. Bronchial dilatations may result from traction on the bronchial walls and may simulate true cavities. The tendency to progressive contraction throughout the fibrotic area causes a marked diminution in size of the lobe. This pulls on the fibrous peribronchitic strands leading to the hilum, which are usually straightened out, like taut wires. If a large part of a lobe is involved, it shrinks to a fraction of its former size. The whole upper lobe may thus come to occupy a small triangular area at one apex. This traction effect upon the trachea is often conspicuous. The trachea is pulled from the midline and arches towards the lesion, often with well-marked angulation. The upward pull upon the hilar structures raises them and the branches of the pulmonary vessels, and the bronchi radiate from a point much higher than usual—occasionally, in extreme cases, from a point just below the sterno-clavicular joint. They descend almost vertically from this point (Figs. 188, 189). The writer has also noted a disassociation as a result of this pull of pulmonary arteries from the bronchi, so that the branches of the artery show an exceptionally clear pure vascular pattern. The effect of the traction reaches as far as the diaphragm, which shows tenting and diminution of movement. Lateral views show involvement of the interlobar pleura, in the form of sclerotic dense lines, with great frequency. An appearance which is sometimes puzzling is that which results from the compensatory emphysema of the opposite lung in cases of complete unilateral fibrosis. A ballooning of the mediastinum occurs towards the fibrosed side. This ballooning does not occur at the posterior mediastinal "weak spot," but is anterior. The anterior edge of the normal lung comes across the midline and invades the opaque side, where it is seen as a soft translucency, with a well-defined convex border. In the lateral view it is manifest that the heart is displaced backwards and separated from the sternum by the emphysematous opposite lung. A similar appearance is seen in cases of non-tuberculous fibrosis.

Massive fibrosis of the whole lung may occur as an end-result of tuberculosis, leading to complete unilateral obscurity. The heart is often pulled entirely to the affected side, leaving the edge of the spine uncovered. The opacity is most marked towards the base, and is largely due to the displaced heart. Considerable thickening of the pleura and elevation and irregularity of the diaphragm are usually present. The intercostal spaces are narrowed, and the capacity of the hemithorax diminished. The condition may be mistaken for bronchial carcinoma causing complete atelectasis.

The oesophagus is often considerably displaced by the fibrosis, and dysphagia may result, as occurred in the case illustrated in Fig. 163. A similar case has also been reported by *Doig*.

CHAPTER XXVIII

TUBERCULOSIS IN CHILDHOOD

AN ATTEMPT to classify the radiographic appearances of a disease showing such protean forms as does tuberculosis of the lungs is only justified under certain limitations. It is necessary to make this attempt in order to simplify the description of those forms which recur with sufficient regularity to constitute easily recognisable groups, and to present a coherent picture of the disease as the radiologist sees it.

The limitations have already been touched upon, and arise mainly from two factors—namely the frequent admixture of different types in the same case, and the very imperfect correlation which is possible at the present day between radiological appearances and clinical manifestations.

PRIMARY TUBERCULOSIS

The appearances seen in young children differ from those seen in adults. The primary lesion in early childhood may be anywhere in the lung and so small as to pass unrecognised in the film. *McPhedran* has shown that a tuberculous focus of $1\frac{1}{2}$ –2 mm. diameter can be shown on a film, but its true nature could not be recognised, and so small a focus might easily be hidden behind the heart or confused with a normal lung marking. There is typically an enlargement of the hilar glands, often massive, which dominates the picture. Often the inflammatory reaction around the primary focus or near the hilum is also massive, producing a widespread shadow. This reaction in the glands in the drainage area of the infection, spreading to neighbouring glands, is the typical feature of tuberculous infection in hitherto uninfected individuals, and occurs almost exclusively in young children.

Pathology

Tubercle bacilli gain access to the lung tissue in most cases through the bronchi. The primary lodgement, which may be anywhere in the lung, and is frequently basal, gives rise to a primary alveolitis (primary focus). Numerous tubercle bacilli are present, quickly surrounded by a wall of small round lymphoid cells. The centre has a pronounced tendency to caseation. There is a rapid tendency to healing by formation of a fibrous capsule and often calcification.

PRIMARY INFILTRATION.—In the lung tissue outside the wall, the alveoli are sometimes filled with an albuminous exudate containing "exudate cells."

with a few small, somewhat eccentric nuclei. The size of this perifocal zone varies. This is the "primary infiltration" (*Redeker*). The disease may either heal or extend. If healing takes place, the primary focus is surrounded by granulation tissue and finally encapsulated. It becomes rounded, and may



FIG. 205.—Pulmonary tuberculosis. Child aged 7. Caseous bronchial glands, with numerous miliary and peribronchial foci. On the outer aspect of the upper lobe is an older caseous lesion, possibly the primary focus. Death from generalised miliary tuberculosis.

calcify later. It often disappears without leaving a trace in the radiogram. The primary infiltration then becomes completely absorbed.

The tuberculous process extends to the lymphatics: tubercles may arise in the lymphatics leading to the hilum. This tuberculous lymphangitis may heal

if the primary focus heals, leaving for a time a fibrous perilymphatic thickening. The tubercle bacilli, on reaching the hilar glands, set up a tuberculous adenitis, which is severe. Caseation usually occurs, and healing takes longer than in the case of the primary lesions in the lung. The younger the child, the more exten-



FIG. 206.—Caseous tuberculous bronchopneumonia in childhood. Numerous caseous foci surrounded by greyish translucent granulation tissue. Intervening lung congested and solid with bronchopneumonia. Death from generalised miliary tuberculosis.

sive, as a rule, the glandular reaction and swelling. The glands may form considerable tumour masses, and if the infection is severe, a periadenitis is also present, most marked in the hilar region, which may extend to interstitial issue, alveoli, or pleura, particularly the interlobar pleura.

On rare occasions it happens that the primary focus does not heal by granulation, but is surrounded by fresh foci by extension, and may thus extend till it reaches the size of a pea, or even of an orange. It may soften, and cavitate (primary cavity). The cavity is at first fuzzy and irregular in shape; later round, and may give rise to bronchopneumonic or miliary foci, or an immediate progressive phthisis.

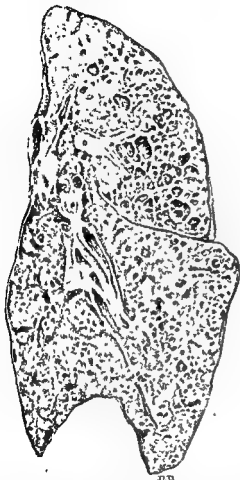


FIG 207. Pulmonary tuberculosis with bronchiectasis. In the lower lobe there is capacious peribronchovascular distribution of tubercles; the affected bronchioles are becoming bronchiectatic. The upper lobe is honeycombed with larger bronchiectatic cavities, apparently of similar origin. From a child, aged 1 year.

Ranke's Classification

Ranke attempted to divide the course of tuberculosis into three stages by analogy with syphilis. He differentiates the stages as follows:

(1) The initial pulmonary focus, constituting, together with the glands, the "primary complex."

(2) The secondary stage of generalisation with development of hypersensitiveness and haematogenous spread (miliary tuberculosis).

(3) A tertiary stage with high immunity and isolated tuberculosis in the lung (adult type).

THE PRIMARY COMPLEX.—The pathological nature and development of this have just been described.

THE SECONDARY STAGE.—The bacilli may reach the blood-stream either from the lung lesion or more often from the lymph nodes, and general infection occurs, most commonly in childhood. In its severest form this is a miliary tuberculosis of the classical type; in its less severe form there are transitory periods of fever with lesions scattered throughout the body. This

form of tuberculosis occurs in somewhat older children. The lesions in the lung are mostly exudative, the lymph nodes may react, and caseation and extensive perifocal inflammation take place. In this stage the tuberculin

reaction is, according to *Ranke*, strongly positive. The hæmatogenous form is not always fatal. Much more often disseminate lesions in bone, joints, etc., become chronic and develop into tertiary tuberculosis of the affected organ.

THE TERTIARY STAGE.—Individuals who survive the primary and secondary stages, i.e. the vast majority of infected people, enter the tertiary stage, which is characterised by (1) relative immunity; (2) isolated lesions in a single organ, usually the lung; (3) a productive reaction with granulation tissue and tubercle formation, with little tendency to the non-specific inflammatory reaction known as “epituberculosis”; (4) little tendency to involvement of the lymph nodes. If there are hæmatogenous metastases, they rarely progress, but direct spread to other organs—e.g. larynx and intestines—is common, and miliary tuberculosis may occur as a terminal event. (5) Sensitiveness to tuberculous toxin is decreased, and in spite of the presence of extensive pulmonary lesions with positive sputa, many of the patients show little in the way of toxic symptoms. The cutaneous reaction to tuberculin is often mild in chronic pulmonary tuberculosis cases.

In criticism of *Ranke*'s classification, *Fishberg* points out that there are numerous limitations of its full acceptance. Often the secondary stage cannot be recognised, and the distinction between the secondary and tertiary manifestations is, as in the case of syphilis, not always practically or even theoretically possible. Many observers believe, therefore, that only *Ranke*'s first stage, the “primary complex,” consisting of a primary focus and glands, is a clearly delimited entity.

The idea that the primary infection in children occurred in the lung parenchyma and that the bronchial and tracheal glands were infected from it is an old one. It was announced by *Parrot* in 1876, who enunciated the rule (*Parrot's law*) that in the child every infection would also affect the glands and that enlarged glands necessarily implied focal infection. *Parrot* also found the primary lesion in autopsies. This work was confirmed by *Küss*, who stated that inhalation of tuberculous bacilli causes a lesion in childhood similar to that produced by subcutaneous inoculation of tuberculosis in guinea-pigs, this form of tuberculosis being at first characterised by a small subpleural tubercle, almost invariably accompanied by infection of its bronchial lymphatic glands. *Küss* found the primary lung focus to be more abundant in the lower lobes. *Albrecht*, from the large material comprising 1,060 autopsies of tuberculous children, confirmed this.

This previous work was brought to notice by *Anton Ghon* in 1916, who made a detailed study of the subject from his own post-mortem material. He showed that the lung focus was nearly always single, often small, and that the glandular enlargement of the hilar and tracheo-bronchial region corresponded with the lesion. For, in the upper lobe lesions, the upper hilar and bronchial glands were enlarged; in lower and middle lobe lesions, the lower hilar and



FIG. 208.—Calcified Ghon's focus at right base. Calcified glands both hila.



FIG. 209.—Lang of a child: healed calcified pulmonary tuberculous, left hilum.

bronchial glands were enlarged. He differed from *Küss* in finding that the upper lobes were more often affected than the lower and the right upper most frequently of all. The radiological evidence of these facts is frequently forthcoming in the form of a calcified focus situated peripherally in a lobe (*Ghon's focus*) with calcification of the corresponding hilar glands. In recent years

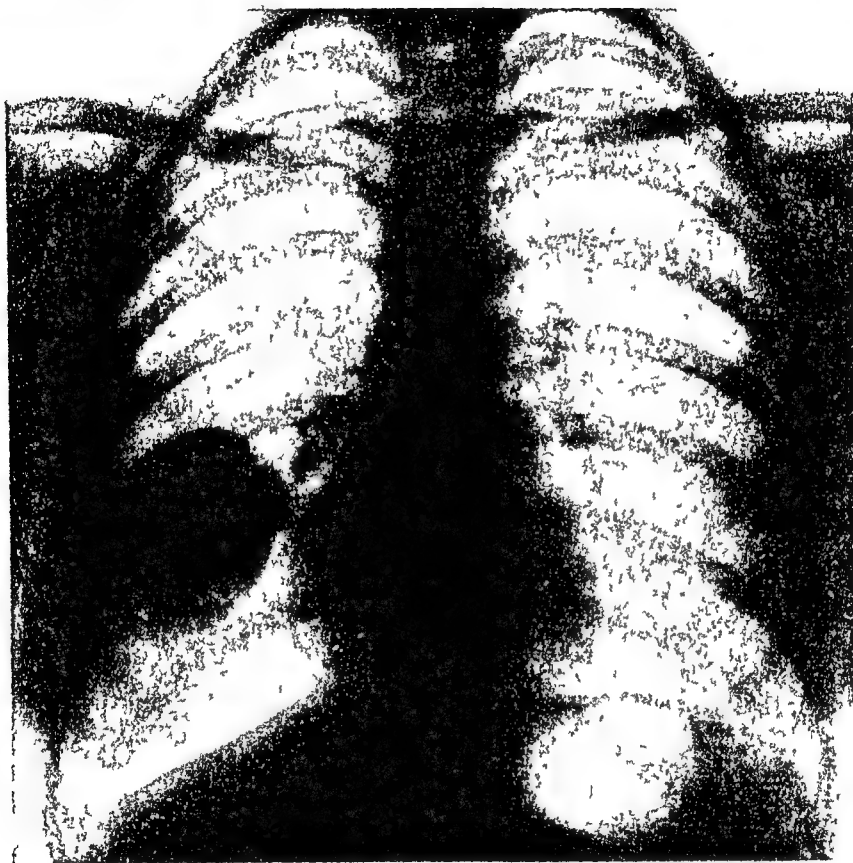


FIG 210.—Calcified tuberculous mass in right lower lobe. F. 29. Under observation for fourteen years. Appearances unchanged. No symptoms during this period

Opie has increased our knowledge of the primary lesion by post-mortem radiographic study of the lungs.

Clinically the primary lesion in children may show different forms. The common complexes are the following (*C. H. Smith*):

(1) Extensive tracheo-bronchial glandular enlargement in infants with fever, failure to gain weight. Radiologically widening of the upper mediastinal shadow, with bulging contours and occasionally obstruction of a bronchus from pressure of the gland.

(2) Marasmic type. Small infants, emaciated, with irregular tempc

Routine X-ray examination may reveal definite tuberculosis in the lymph-nodes, disseminated lung lesions, or even cavities. Prognosis is bad.

(3) *Pneumonic type.* History of fever, prostration, and cough, and clinical signs suggesting consolidation or bronchial pneumonia. X-ray examination shows mottled shadows over part or all of the lung fields (disseminate broncho-pneumonic type) or massive consolidation like lobar pneumonia. In the latter

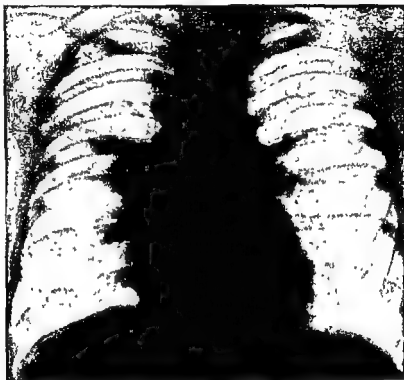


FIG. 211.—Primary tuberculosis in childhood.

case, only the long course, cavitation, or tuberculous bacilli in the sputum, establishes the diagnosis. Recovery is exceptional in this type.

(4) Cases suspected of tuberculosis from the outset and often with tuberculous meningitis, which may be terminal or a first clinical manifestation.

X-ray Appearances of the Primary Complex (Focus and Infiltration)

The shadow of the primary infiltration is at first homogeneous and soft, with ill-defined edges "like a mirror which has been breathed upon." If it subsides, the primary focus may be discerned within it, "emerging like a mountain peak from mist." The larger lesions may show a netlike structure during regression ("induration field"). Gradually the surrounding infiltration

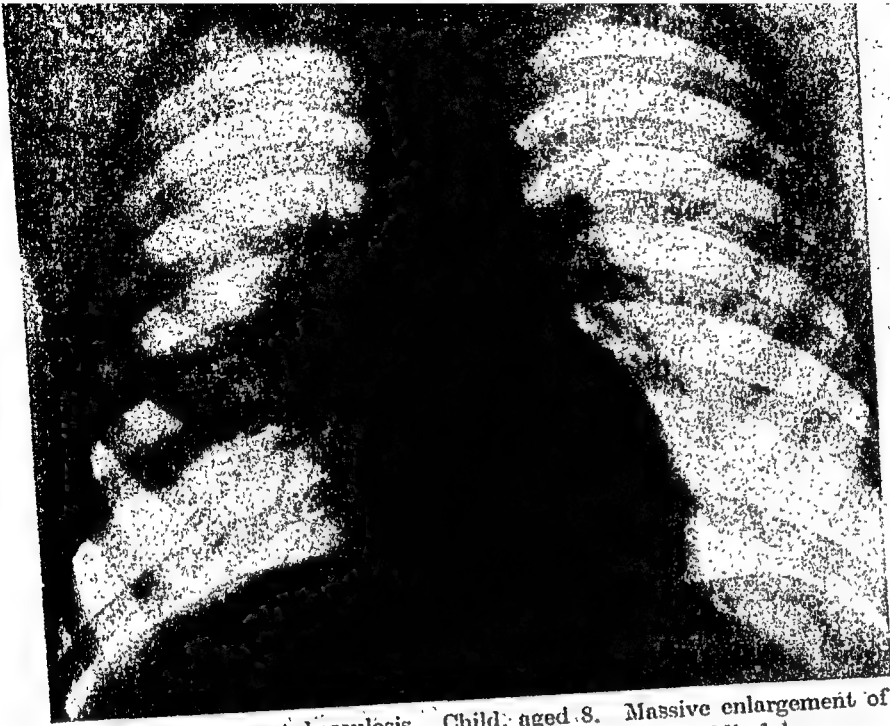


FIG. 212.—Primary tuberculosis. Child, aged 8. Massive enlargement of right hilar glands and exudative lesion in right middle lobe.

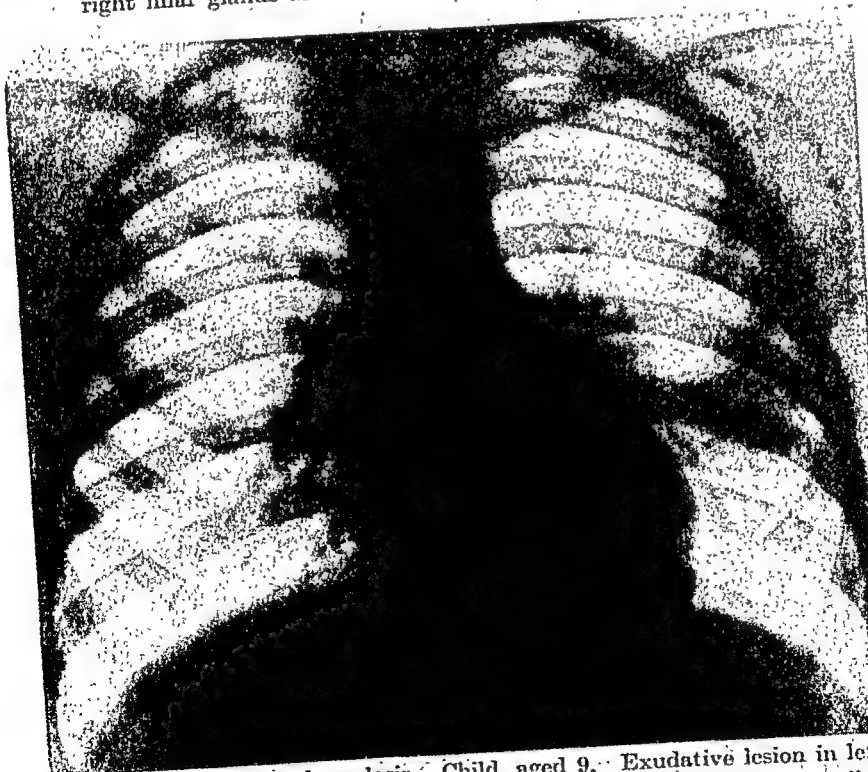


FIG. 213.—Primary tuberculosis. Child, aged 9. Exudative lesion in left lung, with enlargement of corresponding hilar glands—"Bipolar stage."

disappears, leaving only the focus behind. These more extensive lesions may have a lobar distribution, and exactly resemble pneumonia; but whereas pneumonia clears up rapidly, the tuberculous infiltration does so slowly, and may remain unchanged for months. The shadows are frequently triangular, with their base at the periphery, apex towards the hilum. A very characteristic picture is produced by the existence at the same time of a primary lung lesion and the infected hilar glands and surrounding periadenitis. This has been called the "bipolar stage." It consists of two shadows, one in the lung field and one in the hilum, connected by a narrow band of increased density, both triangular with their apices opposed. The hilar triangle may exist alone, if the parenchymatous lesion is small or has cleared up (Sluka's triangle).

Primary infiltrations which last for more than a year form large caseous foci and usually leave behind a calcification of considerable size. Many primary foci occur in situations where they are hidden and never come to light in the radiogram. As a rule only one primary focus is present: more than one occurs only in 4.02 per cent. of all cases. Progression of the primary focus is shown radiographically by increase in size, or appearance of cavitation, which occurs in 25-30 per cent. of infants with this disease. After cavities have formed, a primary phthisis follows. Patchy shadows appear around the cavity, due to caseous foci, which may rapidly extend to form a caseous pneumonia, of lobar bronchopneumonic type.

Tuberculous Bronchial Glands

In the first decade 30-40 per cent. of children show a positive Von Pirquet reaction. The tubercle bacilli are in these cases nearly always present in the perihilar glands. The radiological appearances may take on various forms:

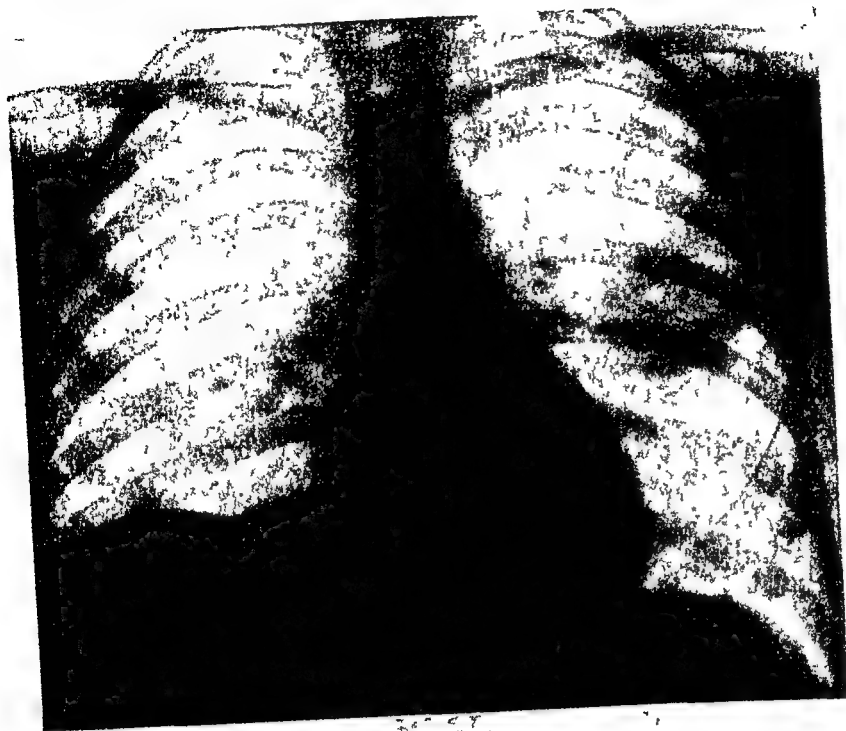
(1) **TUMOROUS FORM.**—The enlarged glands project from the hilum into the lung field, with a convex border which is sometimes lobulated, or, if many glands are fused together, show a nearly straight vertical border. Later calcification appears in the glands, and persists throughout adult life. It is a matter of everyday experience to find these glands, though calcified primary lung foci are encountered somewhat less frequently.

(2) **INFLAMMATORY FORM.**—If the glands are surrounded by secondary inflammatory changes, periadenitis, a triangular soft shadow, extending outwards with its apex in the lung, is formed. This shadow conceals the glandular enlargement. Examination in the hollow-back position sometimes shows this infiltration to extend outwards into the lung along the main interlobar fissure.

Hilitis.—A more diffuse form of hilitis occurs in which a shadow covers the whole hilar region, fading out into the lung tissue with increase in the peripheral bronchial and perivascular shadows at its edge.

(3) **THE INFECTION MAY SPREAD TO THE INTERLOBAR OR MEDIASTINAL PLEURA**, which become visibly thickened. The special form described by *Fleischner* as mediastino-interlobar pleurisy has been traced by him to direct

FIG. 214.—Acute tuberculous bronchopneumonia. Child, aged 13.



Same case 10 days later. Cavitation of the lesion in the left lung.
Extension of the bronchopneumonia at right base.

infection from a gland lying near the hilar end of the lower part of the main interlobar fissure, from which it is separated only by a thin layer of pleura.

Epituberculosis

The massive shadows, often involving an entire lobe, which are to be found in children suffering from tuberculosis, are extremely interesting, and their exact nature is still uncertain. The name "epituberculosis," given to them by *Elias* and *Neuland*, denotes a non-specific exudative reaction, a pneumon-

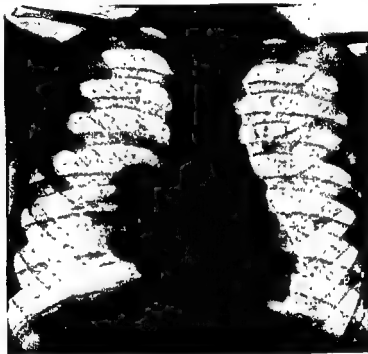


FIG. 215.—Pulmonary tuberculosis. Boy, aged 8. Marked enlargement of hilar glands. Triangular infiltration of middle lobe (Sluka's triangle.)

itis. Epituberculosis may occur as soon as the tissues have become allergic, a few weeks after the original infection. Some consider it to be analogous to the excessive dermal reaction which occurs after subcutaneous injection of tuberculin in a patient sensitised by previous infection. *Parsons*, however, doubts this, pointing out that it is not transient like the skin reaction, but may persist for months, or even years. *Parsons* and *Spence* have shown that active bacilli can be obtained from the "epituberculous" areas by puncture.

Clinically it is unlike pneumonia, being rarely accompanied by acute symptoms. Radiologically the epituberculous shadow is often lobar, with a

FIG. 216.—Epituberculosis.
Female child, aged 11 months.
Contact case. Mantoux +.
Onset five days previously.

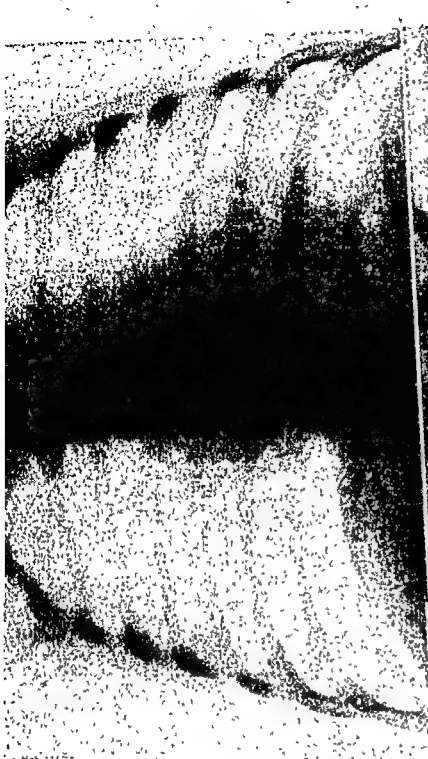


FIG. 218.—Same case two months later. The whole right upper lobe now consolidated. Death occurred from tuberculous meningitis.



FIG. 217.—Same case two weeks later. Consolidation is now present in the base of the right upper lobe.

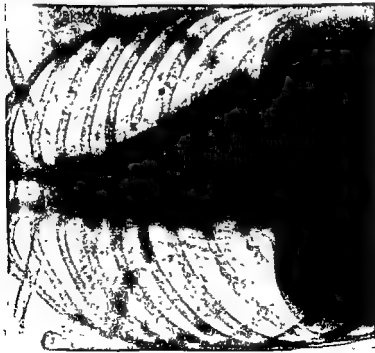


FIG. 219.—Pulmonary tuberculous in a boy aged 12 Infiltration of right upper lobe Enlarged hilar glands.

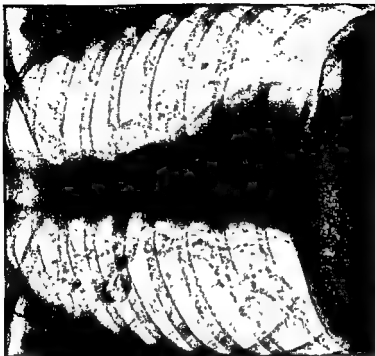


FIG. 220.—Same case 1½ years later Healed with calcification of the pulmonary lesion and glands.

predilection for the upper lobes. It resolves slowly, clearing up first at the periphery. In some cases there is a concomitant atelectasis, indicated by displacement or concavity of an interlobar fissure. It has been suggested that this atelectasis results from pressure of enlarged hilar glands upon the bronchi, but it may occur in the absence of any demonstrable glandular enlargement. During the stage of resolution the shadow may present an exactly similar picture to that of the triangular or flame-shaped shadow of the hilar "periadenitis" or hilar pneumonitis, named by *Burton Wood* the "hilar flare." Conversely a hilar periadenitis may extend, and finally occupy an entire lobe. It is therefore clear that only by repeated X-ray examination, combined with a careful clinical follow-up, will it be possible to arrive at a definite conclusion.

Apical Infiltration

Occasionally in childhood lesions of the adult type can be found. The early apical lesion is relatively rarely encountered in very young children, but as the age of puberty is neared, apical lesions of the adult type become more frequent. In children, as in the case of adults, it is doubtful whether this apical lesion is the result of an endogenous reinfection or of an exogenous infection. As is the case in adults, this form in children rarely spreads from a small apical lesion, but the patient comes with apparently abrupt onset of a lesion of considerable extent. The shadow is due to perifocal inflammatory change around the tuberculous focus. It is usually infraclavicular. Cavitation occurs in a high percentage of cases. Clinically the severity of the symptoms varies. Symptoms may be slight and rapidly disappear, suggesting an influenzal attack. After a free interval, during which cavitation occurs, with phthisical extension, the child may be brought for examination suffering from active phthisis. In favourable cases the early apical infiltration may disappear and leave only slight fibrotic changes at the apex. In unfavourable cases the disease extends by aspiration either in the form of a tuberculous bronchopneumonia, or the appearance of fresh foci in the same or other lung ("daughter infiltrates").

Chronic tuberculosis at school age infects the upper lobe almost invariably. Lesions in the lower lobes are not to be considered tuberculous unless there are symptoms of toxæmia and positive sputum.

CHAPTER XXIX

MILIARY TUBERCULOSIS

THE DISSEMINATION of tubercle bacilli through the blood-stream may follow rupture of a caseous gland into the blood-vessel, or occur in other ways. It is

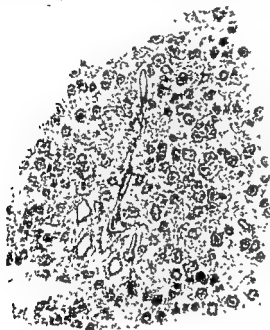


FIG. 221.—Miliary tuberculosis. 4 Section of child's lung. The average diameter of each nodule is $\frac{1}{8}$ inch. Each consists of two or more "tubercles" having classical zones of round cells, epithelioid cells, and giant cells, a radiogram would show innumerable shadows of pin's head size, separated by minute patches of air containing lung, often appearing to coalesce by overlap; the more superficial or more isolated nodules would be individually recognisable.

possible that this dissemination will only lead to the development of miliary tuberculosis under certain conditions of resistance of which we know little. It is, at all events, certain that hæmatogenous dissemination with the implantation of a few or even many colonies of tubercle bacilli in the lung tissue does not invariably evoke the serious clinical picture usually associated with the name "miliary tuberculosis," and that there exist many cases of blood-disseminated tuberculosis of the lungs in which the prognosis is relatively favourable. The writer has met with ambulant cases of disseminated tuberculosis in children, and which were clearly not bronchopneumonic in origin, with relatively mild symptoms. Our knowledge of this condition has been increased by routine examination of school children carried out abroad, which has brought many such cases to light. Redeker states, as a result of a large number of such examinations, that acute miliary tuberculosis, in the old sense, forms but a small selective group of the hæmatogenous forms of tuberculosis.



FIG. 222.—Acute miliary tuberculosis. M. 27. One year previously spontaneous pneumo-
thorax, with negative sputum. Continued work against advice. Three weeks later,
illness. Sputum positive. Death from miliary tuberculosis.

Radiological Appearances

ACUTE MILIARY TUBERCULOSIS (GENERALISED TYPE).—The lung fields are uniformly studded by innumerable dots of opacity, varying in size from a pin's head to 2 or 3 mm. in width. The distribution is universal, and the apices do not escape. The normal lung markings tend to disappear and may become

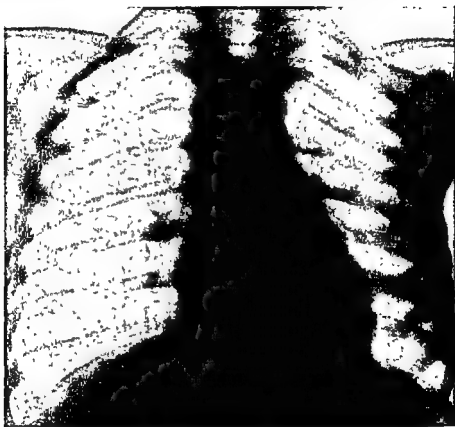


FIG. 223.—Miliary tuberculosis

invisible. The individual shadows are due to the areas of reaction surrounding the microscopic tubercles. They are rarely visible on screen examination; the fluoroscopic picture is one of uniform diminution in translucency, with poor air entry and diaphragm movement, and the eye obtains an impression that the shadow is slightly granular. A good radiogram will show all the lesions which lie in the part of the chest nearest to the film. These cases are part of a generalised miliary disease, and are often associated with tuberculous meningitis. Recovery is almost unknown. *Liebmann* has, however, observed a case of a child with an indubitable miliary tuberculosis of the lung of the fine dis-

seminate type, who became afebrile and left the clinic after some weeks, apparently healed. Not until six months later did a fatal relapse occur.

CHRONIC MILIARY TUBERCULOSIS.—The occasional accidental discovery of calcified disseminate lung foci of miliary distribution, with no predilection for the bases, has for some time been held by many radiologists to be evidence that a chronic miliary form of tuberculosis exists, from which the patient recovers. As already mentioned, there is evidence that this does occur (*Pilger, Blaine,*

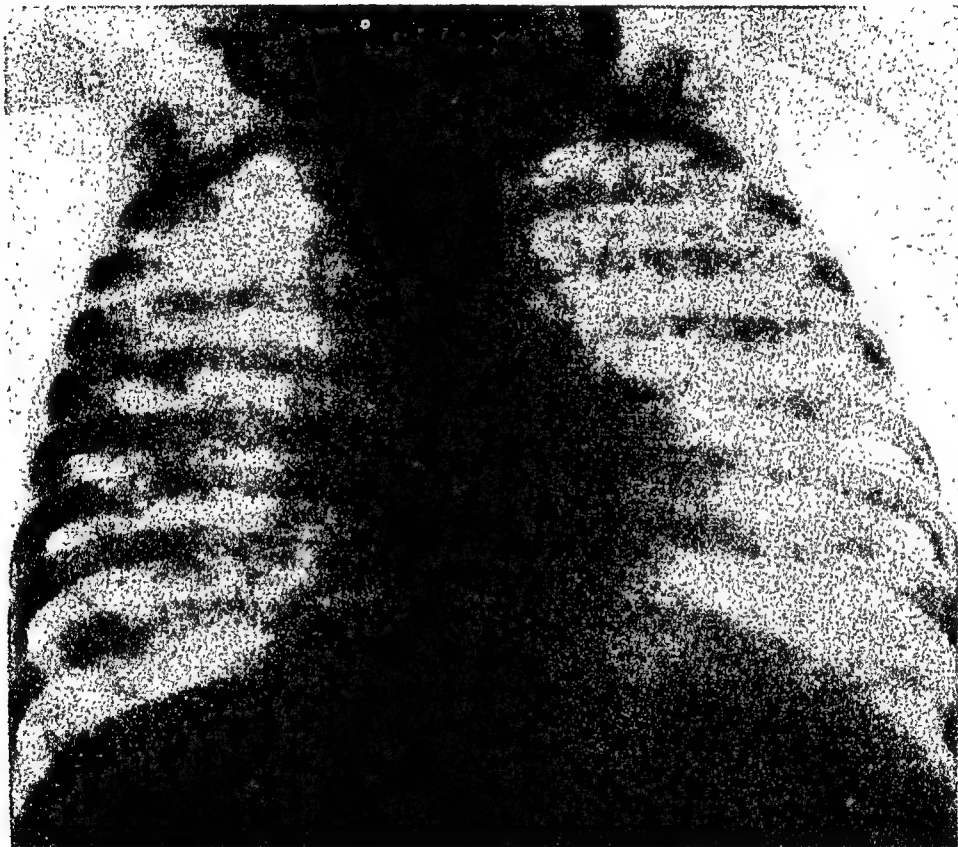


FIG. 224.—Terminal stage of miliary tuberculosis. The child had been ill for several months. Death two days later. Post-mortem: Generalised miliary tuberculosis.

Dunham and Shavelem, Opie). *Assmann* has separated hæmatogenous miliary tuberculosis from disseminate peribronchitic forms, and the work of *Simon, Braüning, Redeker*, and others has enabled a distinction to be made between an acute hæmatogenous pulmonary dissemination and its sequelæ, and the better known clinical picture of miliary tuberculosis. Such dissemination may occur frequently, either immediately following development of an early lesion, or in children with infected bronchial glands. It occurs near the age of puberty with special frequency, and occasionally in the adult. Four radiological

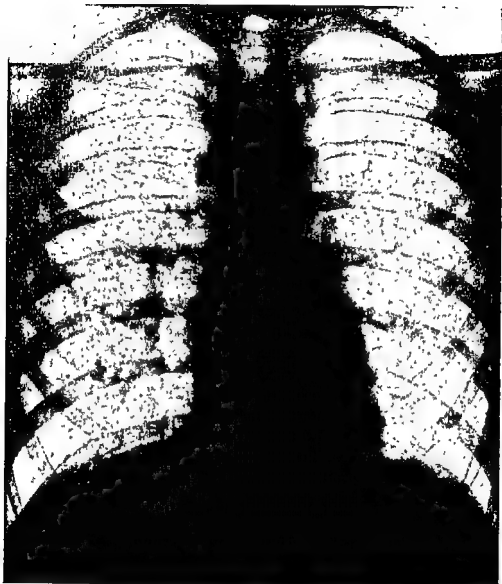


FIG. 225.—Chronic miliary tuberculosis in a girl of 14. Spleen and lymphatic glands enlarged. The pulmonary infiltrations gradually disappeared during the following year.

stages are described. (1) An early hyperæmia and congestion of the pulmonary vessels, resembling a "back pressure" lung. (2) In a few days, or weeks, the appearance of visible foci, some round, others ill-defined and irregular. The distribution is universal, or more marked in the upper lobes, coarse and less uniform than the classical miliary type. The shadows are due to a perifocal

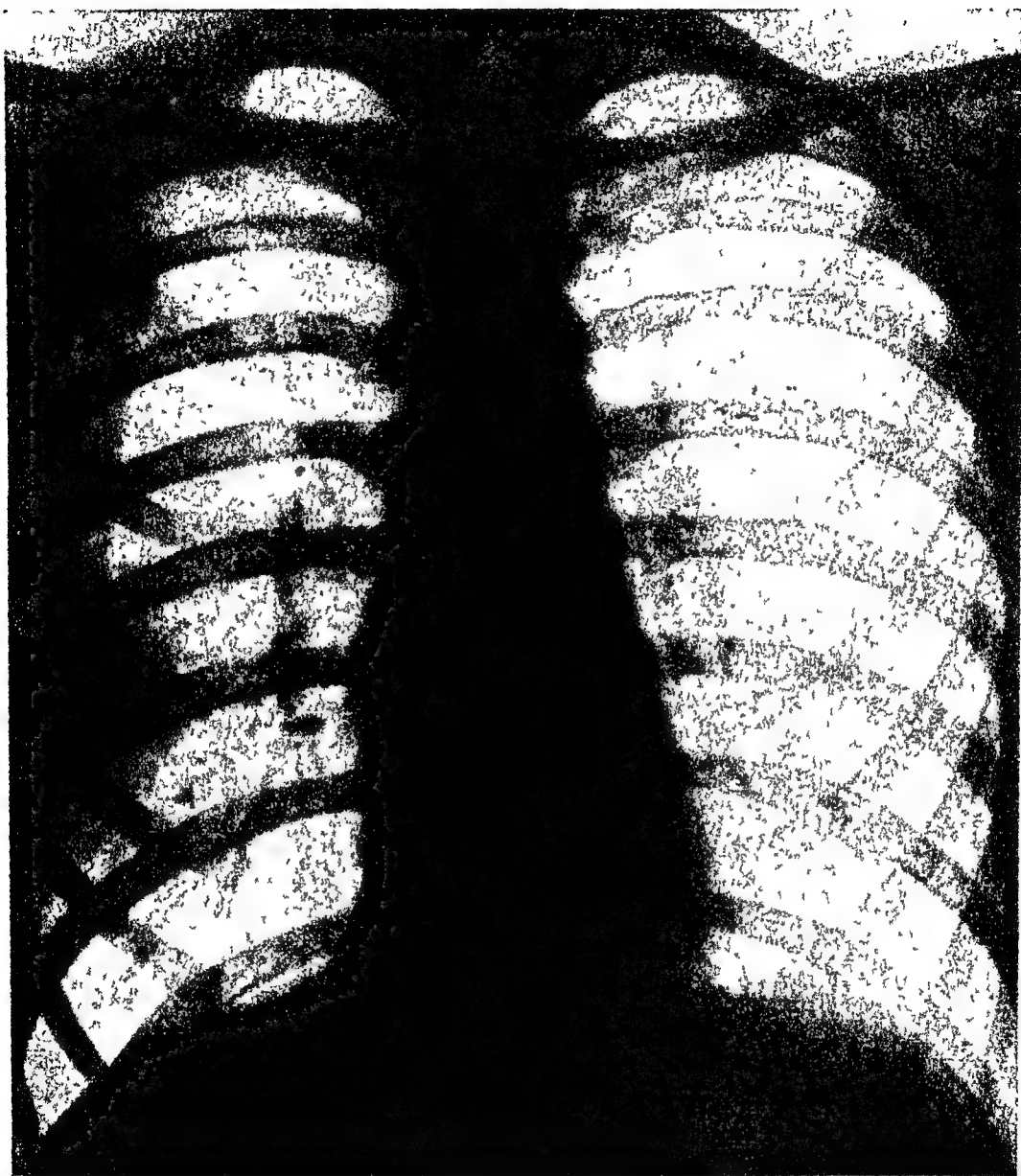


FIG. 226.—Chronic miliary tuberculosis. Same case as Fig. 225 taken three years later. This shows only a few residual stipplings at the apices. Patient in good health.

reaction. (3) In the third stage the perifocal reaction subsides and the individual lesions become more distinct. The vascular shadows diminish, but are still clearly defined. (4) In the fourth stage some lesions disappear, others remain, with sharper and better defined contours. Scar formation leads to coarse cobwebby reticular pattern, with scattered foci, at considerable intervals.

which gradually become hard, fibrotic, or calcified. The clinical course is often benign, in striking contrast to the radiological picture. A case of this nature is reported by *Sante*.

The most recent and complete account of chronic miliary tuberculosis hitherto published is that of *Hoyle and Vaizey*.

They present ten personal cases and have collected 100 from the literature. The condition is most often found in adolescence or early adult life. A

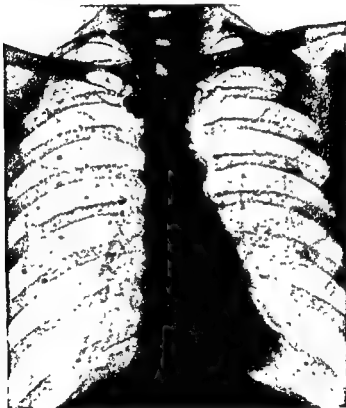


FIG. 227. —Calcified miliary tuberculosis.

cardinal feature of the disease is the disproportion between the clinical signs and the X-ray findings. The disease is latent. The sputum is often negative and the Mantoux reaction may be negative. About one-third of the cases have other lesions, splenomegaly, diffuse lymphadenitis, or tuberculous lesions of the genito-urinary tract, bone, or joints.

The radiological picture is similar to that of acute miliary tuberculosis; the lesions may be of the fine miliary or coarse miliary type. A rare form has an associated tuberculous lymphangitis; in this form the discrete lesions may

be marked by a fine reticular pattern of linear shadows radiating from the hila through the lung fields; this pattern has been proved at autopsy to be due to tuberculosis of the peribronchial and perivascular lymphatics, and in this form dissemination occurs by the lymphatics.

Death may occur within a year, but some cases have been followed to recovery, with or without residual tuberculous lesions.

The case illustrated in Figs. 225 and 226, under the writer's observation for over three years, illustrates complete recovery with some small residual scars in the apices. F. 14. Listlessness, loss of weight, enlarged glands in neck and axillæ, enlarged spleen. Both lungs studded with coarse miliary infiltrations. No cough or sputum. After six months the lesions began to clear and gradually disappeared in a year, except for residual scars at the apices, which have remained stationary. The child is now 17 years of age, backward physically and mentally, but in good general health and gaining weight. No splenic or glandular enlargement. Mantoux feebly positive (1 : 100).

Calcified miliary lung lesions were found by *Sayers* and *Meriwether* in 125 cases during routine examination of 18,000 mine-workers. Over half of the patients were symptomless. The lesions were scattered through the lungs, but most numerous at the bases. Tubercle bacilli were rarely found.

Sutherland has reported thirty-eight cases of miliary calcification in the lungs in rural population.

Hegler and *Holthusen* have described nine cases of miliary tuberculosis, two of which became completely cured, while some others ran a chronic course.

It is believed by *Simon* that the small rounded calcified foci occasionally met with in the supraclavicular apex are residual lesions from a hæmatogenous spread, the other lesions having become entirely absorbed.

Differential Diagnosis of Miliary Tuberculosis

PNEUMOCONIOSIS.—The classical form of miliary tuberculosis with fine nodules could hardly be compared with pneumoconiosis, but the coarser forms may closely resemble pneumoconiosis of the rapidly developing type, during the nodular stage. The distribution, density, and size of the pneumoconiotic nodules are, however, in the writer's experience, less uniform. The nodules are first found peripherally, especially in the axillary borders of the upper lobes. They are harder and better defined than those of miliary tuberculosis, and commonly combined with emphysema. In the third stage the coarse fibrosis and the confluence of the shadows is quite unlike anything seen in miliary tuberculosis.

BRONCHIOLITIS, in the course of measles or influenza, produces a picture resembling miliary tuberculosis, but the lesions are, as a rule, coarser and less well defined, and tend to become confluent. *Assmann* has seen a case of bronchiolitis obliterans, in which the post-mortem findings so closely resembled miliary tuberculosis that it was so described until histological investigation

disproved the diagnosis. This condition is an end-result of influenzal bronchiolitis or of irritative bronchiolitis (gas poisoning), and in it the terminal bronchioli are occupied by fibrous tissue which invades the infundibula, producing a nodular fibrosis. In *Asmann's* case the apices were not appreciably involved in the radiogram.

BRONCHOPNEUMONIA.—The predominant affection of the bases is, as a rule, sufficient to distinguish it from miliary tuberculosis.

OTHER CONDITIONS.—Miliary carcinomatosis, bronchiolectasis, septicæmic miliary foci, leukæmia or lymphadenomatous disseminate foci, actinomycosis, purpura (*Pape*) and mycotic infections, are other conditions which have at times given rise to difficulty in diagnosis. Bilharziasis of the lungs may simulate miliary tuberculosis (*Mainzer*).

Boeck's sarcoid of the skin is, in 5 per cent. of the cases, associated with miliary lesion of the lung. *Hoyle* and *Faizy* consider that these lesions are, in fact, tuberculous.

MILIARY AMYLOIDOSIS.—*Liebmann* has described miliary amyloidosis of the lungs associated with amyloid disease in other organs. In Xanthomatosis (*Schüller-Christian* syndrome) there may be found a coarse stippling of the lung fields, progressing later to fibrosis, circulatory obstruction in the lungs, and heart failure.

CHAPTER XXX

THE MEDIASTINUM

ANATOMY

THE THORAX, as seen in a lateral radiogram, shows from before backwards three zones which correspond approximately to the three anatomical divisions. Anteriorly is a clear zone behind the sternum, triangular in shape, with its apex downwards; the retrosternal space (anterior mediastinum). Behind this, the shadow of the heart and great vessels (middle mediastinum). Behind this again, between it and the spine, another clear space (posterior mediastinum).

IN THE RETROSTERNAL SPACE lie the anterior margins of both lungs, enclosed in the pleural sacs, the pleura of the two sides being in contact in the upper part of the space. The space also contains the thymus or its remains, an important group of lymphatic glands behind the manubrium (anterior mediastinal glands), and some other glands belonging to the parietal division (see Lymphatic Glands).

THE MIDDLE MEDIASTINUM contains the heart, aorta, and origins of the great vessels of the upper extremity, head, and neck; the pericardium, pulmonary arteries, superior and inferior vena cava, and the vessels of the roots of the lungs with the phrenic nerves passing downwards in front of them, the left phrenic being less closely related to the lung root than the right.

IN THE POSTERIOR CLEAR SPACE are the trachea and the main bronchi entering the lung roots, the lymphatic glands in relation to the air tubes, the œsophagus, the aortic arch and descending aorta, and the vagi. In the upper part of this clear space the lungs of the two sides are nearly in contact, being separated only by the pleura.

In the postero-anterior view, the mediastinum is seen as a shadow with clearly defined contours, except where these are interrupted by the lung roots, and is of a density which varies in its different parts. The structures forming its borders are enumerated elsewhere.

TECHNIQUE OF RADIOLOGICAL EXAMINATION

The study of the mediastinum is of very great importance, for the detection of lesions in this region can be accomplished by critical radiological examination before there are any reliable physical signs. Differential diagnosis is often a much more difficult matter, but, in general, the radiologist is called upon to assist in differentiating benign and malignant tumours, substernal thyroid, retrosternal abscess, enlargements and tumours of the thymus, enlargem

of the lymphatic glands due to lymphadenoma, lymphosarcoma, leukaemia, inflammation or secondary malignancy, mediastinal abscess, and aneurysms in various situations.

Very rarely can any definite opinion be formed from a plain postero-anterior film, especially if this is of the "soft" variety. Nor will a "routine antero-posterior and lateral" suffice. The whole mediastinum must be scrutinised on the screen from different angles, films being taken in those positions which show the lesion most clearly. Attention is directed to the following points in particular:

- (1) The size of the lesion, with measurements.
- (2) The shape and definition of the edge, whether sharply defined or indefinite. If the edge is indefinite, whether this is due to actual invasion of the lung through the pleura, or whether it is due to compression and condensation of the lung detail or congestion of vessels of the lung from pressure on the lung root. The shape of the edge, whether smoothly rounded or lobulated, or obviously formed by the fusion of several masses.
- (3) The site of the lesion: whether arising from the uppermost part of the mediastinum, and if so whether it is continued upwards into the neck; whether the lower edge of the shadow involves the lung root, or stops short of the hilum; and in particular whether the shadow is mainly in the anterior, middle, or posterior regions.
- (4) The exact relations of the shadow to the aorta, the heart, and the pericardium; whether it pulsates, and if so whether this is expansile or communicated; whether there is displacement of the trachea or oesophagus.
- (5) Whether the lesion is unilateral or bilateral.
- (6) Whether there is movement of the lesion on swallowing.
- (7) Whether there is bony involvement of ribs, spine, or sternum.
- (8) Enlargement of lymphatic glands elsewhere in the body should be looked for.

MEDIASTINAL TUMOURS

Primary Mediastinal Tumours may arise in various tissues:

- (1) From lymphatic glands: Lymphadenoma, lymphosarcoma, lymphoma (leukaemia).
- (2) From the thyroid gland: Substernal thyroid, simple or malignant.
- (3) From the thymus: Simple hyperplasia, thymic cyst, thymic tumour.
- (4) From connective tissue: Lipoma, fibroma, xanthoma, chondroma, chondrosarcoma, and sarcoma.
- (5) From pleura: Endothelioma.
- (6) From nerve tissue: Ganglioneuroma, neurofibroma, "hour-glass tumour," neuroblastoma.
- (7) From embryonic elements:
 - (a) Vestigial. Tracheobronchial cysts.
 - (b) Dermoids and teratomata.

Secondary Tumours of the Mediastinum may arise as a result of metastasis from sarcomata or carcinomata in any part of the body. These are usually blood-borne, but may reach the mediastinal glands from the neck by direct extension through the lymphatic channels which connect the lower deep cervical glands with those of the mediastinum. It is not common for a bronchial carcinoma to present a pure radiological picture of glandular enlargement of the mediastinum, though in a few instances this may be so marked that it dominates the picture. As a rule, the associated hilar and pulmonary changes establish the diagnosis. Direct extension from œsophageal carcinoma may occur, but is rare, and affects the peri-œsophageal lymph glands first.

In general, the mediastinal tumours observed radiologically belong to the lymphoblastoma group, of which the commonest is lymphadenoma. Lymphosarcoma (of the small or large round-cell types) comes next in frequency. Leukæmic tumours and tumours of the thymus are relatively rare, and the benign tumours are exceedingly rare. Of the last-named, dermoid cysts and teratoma supply most of the recorded examples.

The benign tumours are distinguished by their occurrence in younger individuals, by slow growth, comparatively mild symptoms, and complete lack of response to irradiation. Though rare, they are important, since they are often amenable to surgical treatment. It has been emphasised by *Tudor Edwards* that the benign tumours are potentially malignant and that their removal is for this reason essential. He stresses the difficulty of making a diagnosis from a consideration of the clinical signs and radiographic appearances alone, and the need for using all the known accessory methods of diagnosis, including artificial pneumothorax, thoracoscopy, lipiodol injection, bronchoscopy, paracentesis through a wide-bore needle and incision and biopsy.

HODGKIN'S DISEASE

Hodgkin's disease (lymphadenoma : lymphogranulomatosis) is one member of a group of diseases which in American and Continental literature are tending to be classified together under the term lymphoblastoma. The group includes the leukæmias, lymphosarcoma. Hodgkin's disease and sarcomatous Hodgkin's disease.

Primary mediastinal adenopathy is much more commonly due to Hodgkin's disease than to any other member of the group, though it may occur in leukæmia and lymphosarcoma.

Hodgkin's disease is about two and a half times as common in males as in females; intrathoracic involvement is about twice as common in males; sarcomatous Hodgkin's disease is six times as common in males as in females. In a very detailed review of 681 cases of disease of the lymph-hæmopoietic system, including 214 cases of Hodgkin's disease, *Peirce, Jacob, and Hildreth* have correlated the clinical and radiological findings. The series included 198 cases of Hodgkin's disease which had had X-ray examination of the chest. Of

these 37.4 per cent. had normal chests, 37.9 per cent. showed mediastinal or hilar glandular enlargement, 13.6 per cent. infiltration of the lung parenchyma, and 11 per cent. pleural involvement. The high incidence of parenchymatous infiltration is noteworthy. It occurred in rather more than 20 per cent. of those cases with positive chest findings.

Radiological Features

The glandular changes vary in degree, from localised enlargement of the hilar or bifurcation glands to very large tumour masses, but they are nearly



FIG. 228.—Lymphadenoma, involving paratracheal and bronchopulmonary glands. Disappeared after radiotherapy. The discrete shadows in the left upper lobe are lipiodol "leakage". Bulky emphysema at left apex.

always bilateral. In the slighter cases the enlarged glands may be hidden by the heart, and the lateral view is essential to detect them. This view may show glands about the bifurcation of the trachea, or behind the trachea. Enlargement of the retrosternal glands is also common, and is clearly visible in the radiogram as a shadow behind the upper end of the sternum, filling up the space between it and the heart and great vessels. In the postero-anterior view the first shadow is usually seen in the right paratracheal region, sometimes as a slight convexity disturbing the normal contour of the superior vena cava; but it is often bilateral. The upper mediastinum gradually widens. In "hard" films, the glands are often visible as rounded shadows fitting into the con-

cavity of the main bronchi. Tracheal or oesophageal displacement and deformity may be found. The enlargement sometimes remains confined to the upper mediastinum, giving a broad, smoothly conformed shadow, bilateral, and tending to spread upwards to the thoracic inlet. When the tracheobronchial, hilar, and pulmonary glands are involved, and are matted together or overlap, the shadows show a typical lobulated contour (Fig. 228). If they remain discrete, the radiogram shows separate rounded masses in relation to the larger bronchi, and enlarged interbronchial nodes in the perihilar region. Isolated

rounded glands may also be found farther out in the lung field, resembling malignant metastases. In Hodgkin's disease these tend rather to be grouped around the hilum and to diminish more regularly in size from the hilum outwards than blood-borne metastases, which are more universally distributed.

The "mediastinal" type of X-ray picture is twice as common as the "hilar" type.

Parenchymatous involvement of the lung may take many forms :

(a) Radiating strands of peribronchial infiltration, resembling inflammatory disease, tuberculosis, or bronchial carcinoma.

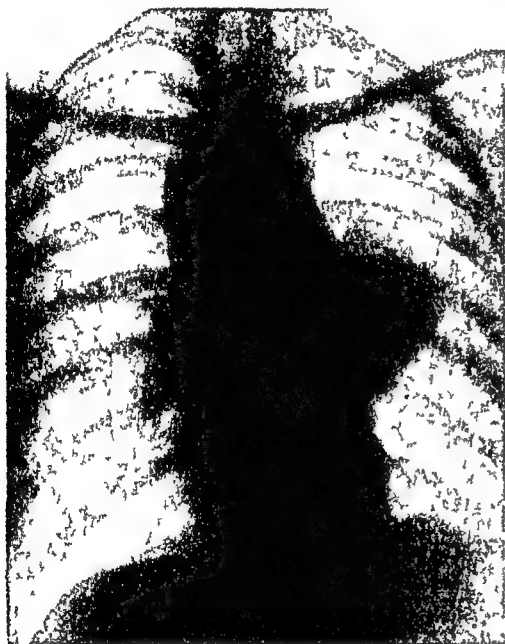


FIG. 229 —Lymphadenoma Right paratracheal and left tracheobronchial glands show massive enlargement

Lateral view Glands of hilum and tracheal bifurcation, and retrosternal glands enlarged.

(b) Coarse rounded nodules resembling metastases, which may in rare instances become cavernous (*Lichtenstein*).

(c) Fine or coarse disseminate masses with ill-defined edges. These cannot always be distinguished from disseminate or even miliary tuberculous lesions, but tend to be more symmetrical and to spare the apices.

(d) Massive pneumonic infiltrations, with or without atelectasis.

A course of radiation treatment may be necessary to clear up the diagnosis, as has been shown by *Desjardins*.

A case of lymphadenomatous infiltration of the lung observed by the writer, proved by the presence of enlarged glands elsewhere, by biopsy of an excised gland, and by the radiotherapeutic test is shown in Fig. 230.



FIG 230.—Lymphadenoma, with diffuse infiltration of lung. M 40 Three months' dyspnea, pain in back, and enlarged glands. Biopsy Lymphadenoma Three months later the pulmonary infiltration had cleared up to a great extent after treatment by radiotherapy



FIG. 231.—Lymphadenoma. Patient, M. 60, has enlarged glands and splenomegaly. The tracheobronchial and bronchopulmonary glands are enlarged, and there is nodular involvement of right lung with interlobar effusion. All lesions disappeared after radiotherapy. Chest normal six months later.

Complications

Fever of the Pel-Ebstein type, pruritus, diffuse lymphoblastomatous infiltration of the skin, laryngeal paralysis, pleurisy and ascites from obstruction of the great vessels, metastasis in bone, direct invasion or erosion of sternum, ribs, or vertebræ, or infiltration of the spinal cord with paraplegia, metastases in brain or testis, and lymphogranulomatous tumour of the gastric mucosa have been observed in Hodgkin's disease. The bone lesions may occur at a distance and are usually osteolytic; an osteoplastic type has been recorded in the spine

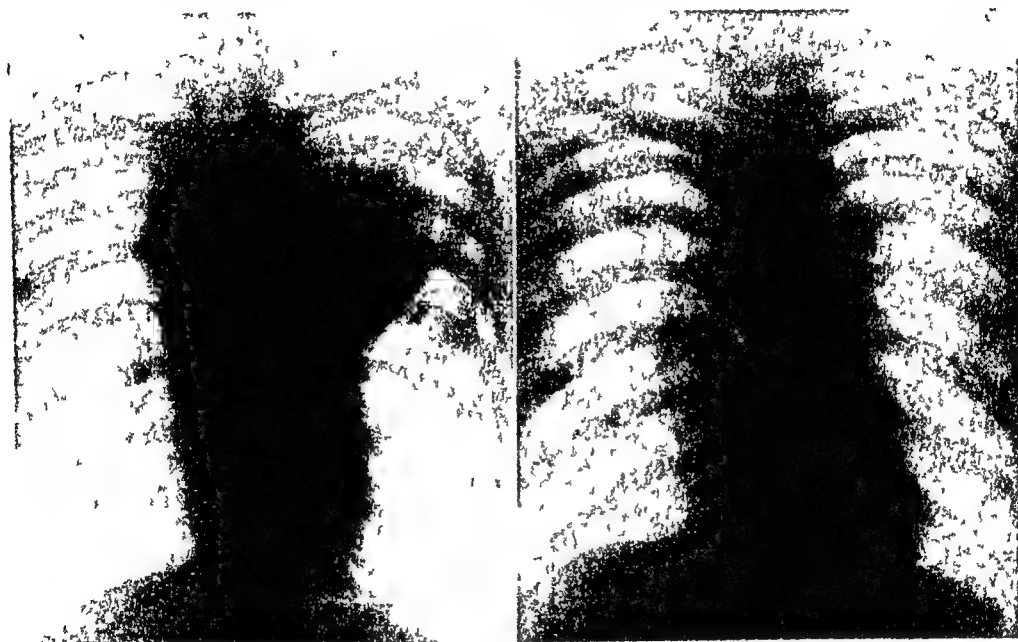


FIG. 232 —Lymphadenoma, M 23. Involvement of left tracheobronchial and retrosternal glands, extension into left upper lobe

Radiogram of same case six months later, following X ray treatment. Slight residual enlargement of the left tracheobronchial glands. A year later there was involvement of left upper lobe

which produces marked increase in the density of one or more vertebræ ("Elfenbeinwirbel"—*Hulten*). According to *Ziegler* (*Schinz*), bone lesions occur in 30–40 per cent. of cases of Hodgkin's disease. The vertebræ, sternum, pelvis, and ribs are most frequently involved, in that order.

Differential Diagnosis

In the majority of cases the presence of enlarged spleen and of glands in neck, axilla, groin, or abdomen, together with the blood-count and clinical course, establish the diagnosis. If these are absent, an exact diagnosis is possible from the X-ray evidence alone. Leukæmia, aleukæmic l,

lymphosarcoma, and thymoma may produce tumour shadows in the upper mediastinum indistinguishable from Hodgkin's disease. The blood picture will differentiate the leukæmias. Lymphosarcomata cannot, as a rule, be differentiated, except by their greater tendency to infiltrate the lung tissue and to present an ill-defined edge. Thymoma tends to invade the thoracic inlet, where it compresses or displaces the trachea. Some thymomata, however, occupy a lower situation. Lymphosarcoma and leukæmic tumours are more strongly radiosensitive than lymphadenoma. Thymoma requires heavier doses of radiation than the preceding, but can be made to regress by X-ray treatment, occasionally with complete cure. Carcinoma is relatively, and the benign tumours completely, insensitive.

The increased mediastinal shadow due to paravertebral abscess of Pott's disease is easily distinguishable from that due to adenopathy on careful examination of the mediastinal space. It lies in the posterior mediastinum. A rare cause of enlargement of mediastinal glands is syphilis. A dermoid is unilateral, and usually projects into the lung field in a manner quite unlike the lobulated, usually bilateral shadow of Hodgkin's disease. The contour of a teratoma is, however, sometimes slightly lobulated or wavy. Substernal thyroid may be unilateral. It rises upwards on swallowing, unless fixed by its great size or by adhesions. Malignant substernal thyroid carcinoma, malignant cervical glands invading the thoracic inlet from the neck, and thymoma all produce a widening of the superior mediastinum and tracheal displacement or compression, resembling that of a simple substernal thyroid, but do not move upwards on swallowing.

In children, tuberculous adenopathy must be considered in the differential diagnosis. In adults, this condition is so rare in the mediastinal and hilar glands that it hardly ever enters into the differential diagnosis. It has been observed in adult native mine-workers in South Africa. It occasionally occurs in this country, and the differential diagnosis is then impossible.

LYMPHOSARCOMA OF THE MEDIASTINUM

Lymphosarcoma arises in the lymph nodes and penetrates the capsule, so that individual nodes are more or less fused. Two forms are recognised histologically: (1) The small-celled lymphocytoma, which is uncommon and almost confined to small children, and (2) The large-celled (reticulum cell sarcoma).

In the mediastinum the tumour grows rapidly, and spreading from one group of glands to another may soon come to fill the entire mediastinal space, and to invade lungs, bronchi, or œsophagus, or perforate the sternum. The symptoms are essentially due to compression, namely dyspnœa, cough, cyanosis, pain, or dysphagia. Laryngeal palsy, with hoarseness or aphonia, or phrenic palsy, may be present; these nerve palsies are more common in lymphosarcoma than in lymphadenoma.

In the later stages there is general weakness, pallor, and loss of weight.

Radiological Appearances

There is a marked increase in breadth of the upper mediastinal shadow, usually bilateral. The outline is smoothly rounded, occasionally wavy or lobulated. As a rule the margins are clearly defined, even though the tumour is massive, for the mediastinal pleura resists invasion and may continue to confine the growth within bounds. It may, however, be invaded, and the tumour, spreading into the lung field, then shows an irregular blurred edge. A certain amount of hairiness of the margin is sometimes due to compression of the adjacent lung and to stasis in the lung vessels which can be seen as prominent linear markings curving upwards from the hilum to the outer side of the mass. These must not be mistaken for "invasion." The strands of shadow quickly disappear as soon as the bulk of the mass is reduced by radiotherapy.

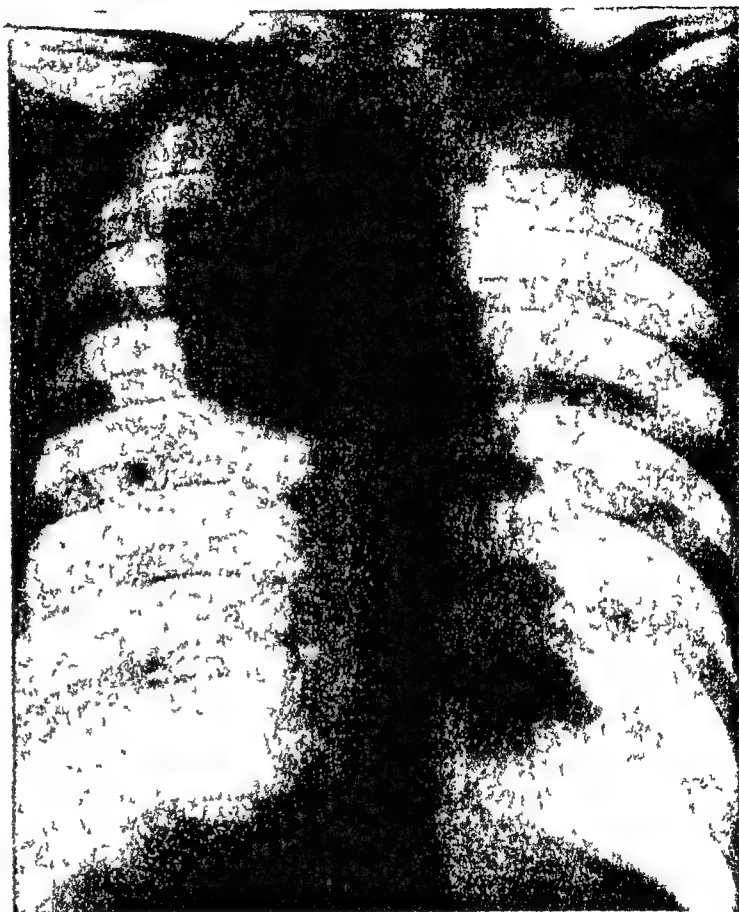


FIG. 233.—Mediastinal tumour, with secondary nodules in lung. M. 59. Recent cough, dyspnoea, and weakness. The mass grew rapidly during two months and the nodules appeared in lung. No primary tumour elsewhere. Death from metastasis in brain. Possibly lymphosarcoma

In the lateral view the growth can often be seen to involve the tracheobronchial and retrosternal glands, and the oblique view will show enlargement of the glands of the bifurcation. If the œsophagus is outlined with barium, the mass may be found to deform it, especially on its anterior contour.

The glandular mass in the upper mediastinum has to be differentiated from aneurysm. This is usually practicable when the glandular involvement is not very extensive, for the aorta can usually be seen clearly through or to one side.

of the tumour shadow ; but the aorta is sometimes completely embedded in tumour, and it is then impossible to make the distinction.

It is stated that lymphosarcoma is more radiosensitive than lymphadenoma. This is possible in view of its activity and rapid growth, but the writer has found fairly wide variations in the sensitivity of lymphadenoma itself, and does not consider that any reliable deductions can be drawn from this test in differentiating the

two conditions from one another, though both may usually be distinguished by the radiotherapeutic test from carcinoma, which is much less sensitive.

Thymoma, which produces a tumour very similar in its appearances to lymphosarcoma, is about equally radiosensitive.

SUBSTERNAL THYROID

Substernal goitre is usually a downward prolongation or outgrowth of a cervical goitre. It may, however, be developed within the thorax, and be unaccompanied by any obvious thyroid enlargement in the neck. It is very

frequently unilateral. In old age, with increasing kyphosis, a long-standing goitre of the neck may become less obvious, but X-ray examination may show it to have become retrosternal. A toxic thyroid sometimes has a retrosternal extension, or is entirely retrosternal. A substernal goitre usually displaces the aorta downwards, and the innominate and subclavian arteries outwards to either side, causing a broadening of the mediastinal shadow which is



FIG 234 — Calcified thyroid adenoma Both lobes densely calcified Trachea compressed

often asymmetrical and frequently unilateral. The aortic knob becomes prominent, as a result of the downward displacement of the arch. The contour of the enlarged substernal thyroid, traced upwards, is continuous with that of the cervical enlargement. The usual picture is that of a somewhat pyramidal shadow, with the narrower end downwards, the trachea is compressed laterally; the greatest narrowing is seen at, or a little below, the level of the clavicles; but it is usually visible also in the neck, a point of distinction from tumours arising in the mediastinum. For alterations of the cardiac contour in toxic thyroid enlargements see the section on the heart. Sometimes, when the

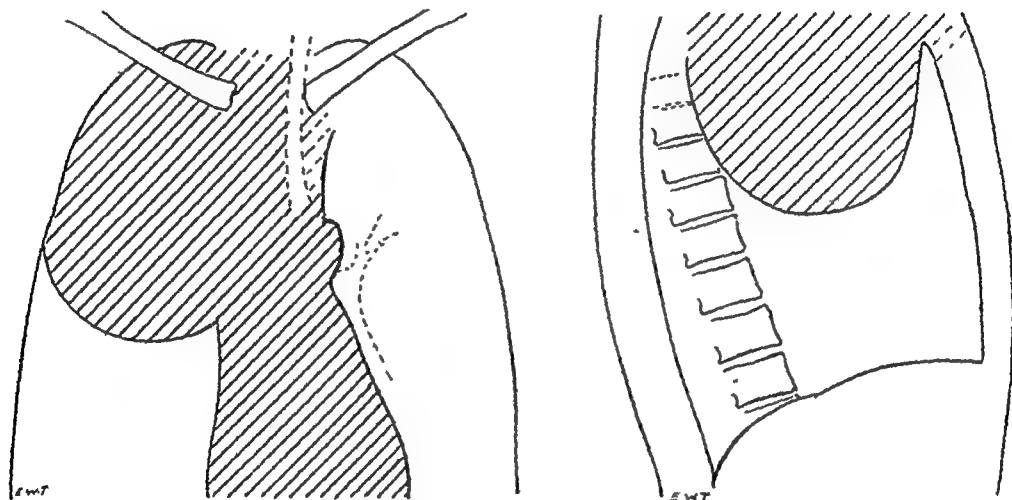


FIG. 235.—Diagram: Large, unilateral substernal thyroid—after Chaoul.

thyroid enlargement is nodular, two or more distinct indentations of the trachea may be observed.

A substernal thyroid usually moves upwards quite distinctly with swallowing, unless malignant or adherent.

The right oblique view shows a shadow passing downwards in front of and somewhat overlapping the trachea. In full lateral view backward displacement of the trachea and some indentation of its anterior margin may be observed.

Calcification may be visible in the cervical or substernal part of the thyroid. It may be circumscribed, or disseminate and "flocculent."

DIFFERENTIAL DIAGNOSIS is from aneurysm of the aortic arch, or from malignant glands or tumour. Enlargement of the upper mediastinal shadow has been observed by the writer in a case of malignant glands in the neck with extension downwards into the superior mediastinum, and in a case of thymoma with upward extension, verified by post-mortem.

The principal points differentiating substernal thyroid from aneurysm of the arch are as follows:

- (1) Aneurysm usually displaces the aortic knob upwards or outwards.

large thyroid tumour displaces it downwards, but a small one does not. If, however, the aneurysm arises from the upper surface of the arch, the aortic arch may be displaced downwards.

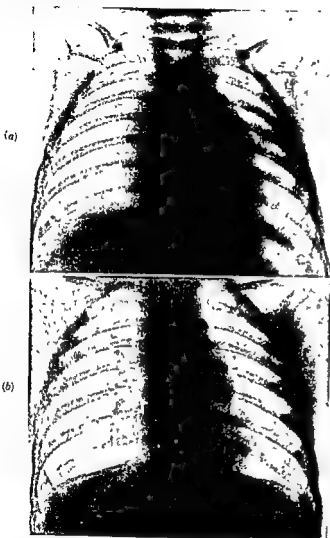


FIG. 236.—Normal infant. (a) taken on expiration, (b) taken on inspiration. Effect of respiration on mediastinal shadow. The dilatation of the superior vena cava on expiration simulates an enlarged thymus.

(2) Lateral compression of the trachea is very rarely caused by aneurysm; displacement, not compression, is the rule.

(3) The calcification in aneurysm is in the wall of the sac, usually curvilinear, never sharply circumscribed or flocculent.

(4) Pulsation is much more common in aneurysm.

(5) Aneurysm does not extend upwards into the neck.

(6) Displacement and indentation of the œsophagus is the rule in aneurysm, and exceedingly rare in goitre.

(7) Upward movement on swallowing is the rule in substernal thyroid, but can only occur in aneurysm if it is adherent to a bronchus or the trachea.

A very large unilateral substernal thyroid may simulate a benign lung tumour. Such a case has been published by *Chaoul*; it filled the

upper part of the right lung field. The trachea was displaced, but the aorta and the great vessels were undisturbed. A diagram of this tumour is given (see Fig. 235).

THE THYMUS GLAND

Hyperplasia of the Thymus

Simple enlargement of the thymus gland in children has been studied radiologically in America more than in this country. Readers are referred to the works of *Pancoast* and *Pendergrass* and of *Donaldson*.

Clinically there may be dyspnoea, attacks of cyanosis with unconsciousness and "thymic stridor," but symptoms may be absent in many of the cases

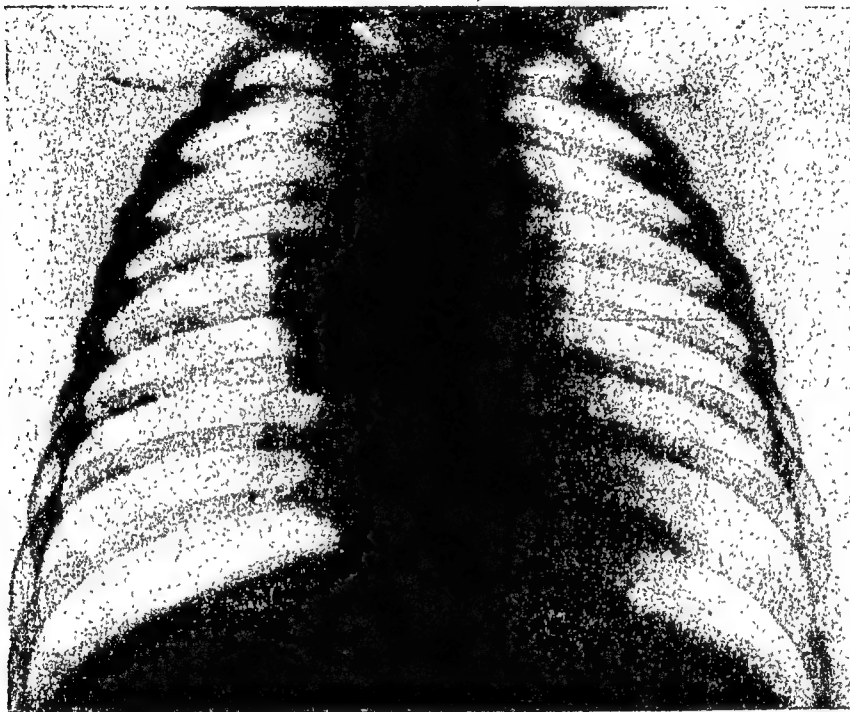


FIG. 237.—Enlarged thymus. M. æt. 1 year.

which show an enlarged thymus. There is a tendency for the condition to be familial.

X-ray examination may show a broad convex shadow in the upper part of the mediastinum, wider below and sometimes fitting over the upper part of the heart shadow like a cap (*Hochsinger*). It changes shape with respiratory effort. Minor degrees of enlargement are difficult to distinguish from that due to a distended superior vena cava, which also becomes enlarged in infants during crying or struggling (Fig. 236). In the lateral view the trachea shows a peculiar kinking due to the thymus at the thoracic inlet (*Pancoast*).

Radiological evidence is not always reliable. Various writers have recorded that autopsy has often failed to show any thymic enlargements.

children with positive X-ray findings, or, conversely, has shown thymic enlargement to be present with no X-ray signs. *Hochsinger's* observations have, however, been confirmed by others upon occasion, notably by *Zuckerlandl*. Broadening of the upper mediastinum was marked in some of the latter's cases, and disappeared after X-ray therapy. Enlarged thymus in adults is very rare, but has been recorded by *Assmann* in a case of lymphatic leukaemia. Carcinoma of the thymus and malignant thymoma usually project unilaterally.

RELIABILITY OF X-RAY EVIDENCE OF THYMIC ENLARGEMENT IN INFANTS.—The appended table, after *Donaldson*, shows that in new-born infants there is a general correspondence between thymic symptoms and visible thymic enlargement. Thymic enlargement in Groups Ia and Ib was three times as common in those infants with symptoms as in the unselected group Ia. It is obvious, however, that symptoms often occur with no thymic enlargement.

In older infants, up to 6 months, thymic enlargement appears to persist, and to show the same percentage incidence as in the "new-born, with symptoms", but the symptoms apparently subside in this age period, since the children in this group were not referred as "enlarged thymus" cases, but for routine examination prior to operation for tonsils and adenoids, or for other reasons. After the age of 6 months the incidence of enlarged thymus is very low. The thymus is sometimes enlarged in cases of toxic goitre in adults.

Classification	Total	Positive	Border-line	Negative	%
Group Ia New-born consecutive unselected	500	74	20	397	14.8
Group Ib: New-born with thymic symptoms	166	68	16	81	41.2
Group II: Birth to 6 months, routine chest examination	133	55	6	74	40.7
Group III: Six months to 6 years, routine chest examination	245	11	18	216	4.5

Table: Incidence of thymic enlargement according to Donaldson.

Tumours of the Thymus

Primary tumours of the thymus are rare. *Doub*, in 1930, found reports of about 100 cases in the literature, of which twenty were carcinomata, the remainder sarcomata. Since then they have been reported at the rate of about two a year.

The thymus is a paired organ developed from the third branchial clefts. It has a structure somewhat resembling lymph gland, but possesses a cellular

reticulum of rather large cells, the spaces of which are crowded with leucocytes. The reticulum of the medulla contains cell nests (Hassall's corpuscles).

Primary tumours of the gland may be : (1) Malignant thymoma, or lym-

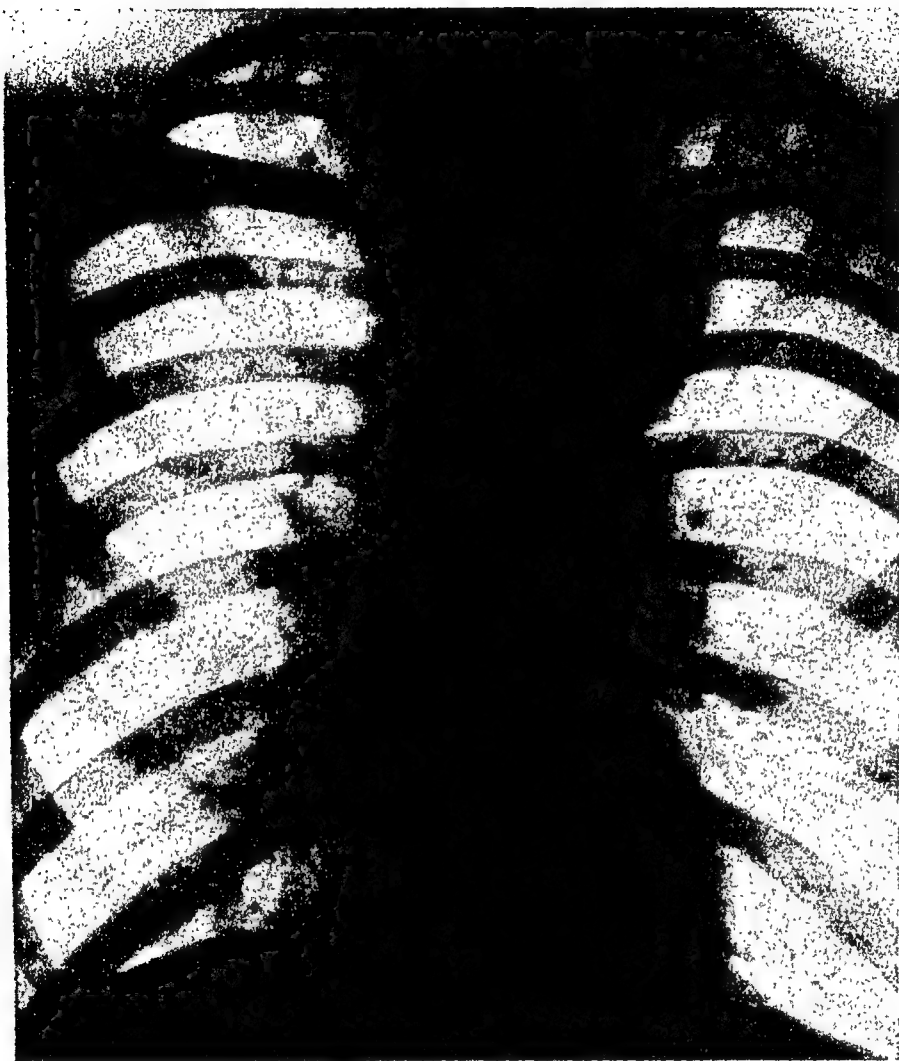


FIG. 233.—Thymoma. M. 22. Clinically there was intense dyspnoea. At post-mortem the growth invaded the superior mediastinum and neck, and the tracheal walls were infiltrated, and in places, perforated.

phosarcoma ; (2) Carcinoma, composed of large cells of epithelial type ; (3) Spindle-cell sarcoma and myxosarcoma derived from the stroma. Sometimes the so-called lymphosarcoma and carcinoma may be mixed, so that the tumour shows reticulum cell areas and aggregations of small lymphoid cells side by side, the structure then being reminiscent of lymph-epithelioma of the

pharyngeal mucosa. Dermoid cysts and simple ciliated cysts may also arise in the thymus.

Thymomata are usually highly malignant and invasive. They occur at any



FIG. 239.—Thymoma. Penetrating film of same case as Fig. 238 to show tracheal compression and displacement.

age. *Doub's* five patients ranged from 25 to 72, but the majority were in the fourth decade.

The post-mortem appearances are thus described by *Doub*: "When the chest is opened at post-mortem a mass is found occupying the region of the thymus, lying in the anterior mediastinum, yellowish or greyish. It may be encapsulated, but is frequently adherent to the sternum and surrounding

organs, and may invade pericardium, lungs, trachea, and glands. Direct invasion is more often seen than distant metastases. The latter may occur to kidney, bone, brain, or other internal organs, and axillary and cervical glands are frequently involved." The clinical findings may be at first those of debility, but pain in the neck and chest is a prominent early symptom. Later the signs are those of any tumour compressing the structures of the thoracic inlet, namely venous congestion, dyspnoea, and dysphagia. In several recorded cases the tumour has invaded and perforated the anterior chest wall. Malignant pleural effusion may occur. Lymphatic leukæmia may be associated, and pulmonary tuberculosis may coexist. Another interesting association is that of myasthenia gravis with thymoma. Two such cases are quoted by *Doub*, and others have since been recorded (*Gold*).

RADIOLOGICAL APPEARANCES.—Thymoma shows a more or less circular sharply defined non-pulsating mass in the anterior mediastinum. It lies below the clavicles, and partly covers the heart. Sometimes it projects more to one side than the other; its position is usually somewhat lower than that of the other mediastinal tumours, which are also more likely to involve the middle and posterior parts of the mediastinum. It is not always possible to differentiate it from them, but a biopsy of an axillary or cervical gland or of the tumour itself may clinch the diagnosis.

In rare instances the tumour may invade the lungs and show a diffuse spreading edge, or still more rarely give rise to multiple metastases resembling miliary tuberculosis.

Thymomata are not amenable to surgical treatment, since they envelop and are intimately bound up with the other mediastinal structures. They are, however, very radiosensitive, and some cases have survived for long periods after X-ray treatment. Treatments of less than 1,000r. delivered in the tumour have resulted in marked regression to about half the original size in six weeks (*Doub*). Complete regression requires heavier treatment or a repetition of the course. Survival periods of as much as six years are known. *Dwyer's* case was alive and well after three years, and one of *Doub's* cases after five years.

RARE MEDIASTINAL AND MIDLINE PARIETAL TUMOURS

Neurinomata and ganglioneuromata are tumours arising from nerve tissue. These may arise from sympathetic, vagus, or intercostal nerves. They cause shadows with clearly defined contours which project from mediastinum or thoracic wall into the lung field (1) and (2). *Assmann* illustrates a large mediastinal tumour due to a neurinoma of the vagus.

Neurofibromas form a high percentage of benign tumours. They arise from intercostal nerves; nearly always in the upper posterior mediastinum, but may occur on the chest wall. They may become malignant.

Histologically they show a structure similar to that of acoustic neuroma.

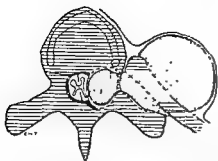


FIG. 240 — Diagram: Hour glass (dumb-bell) neurofibroma. The intraspinal part compresses the cord; the extraspinal portion elevates the pleura, enlarges the intervertebral foramen, and erodes the vertebral body, transverse process, and ribs.

Palisade arrangement of the nuclei may be seen. A few have the structure of xanthoma. Malignant degeneration may occur.

SYMPTOMS are mild or absent: when large these tumours may cause dyspnoea or irritable cough from pressure on the trachea, or dysphagia by pressure on the œsophagus. Pain along an intercostal nerve is rather frequent; it increases in severity if malignant changes supervene; occasionally Horner's syndrome (from involvement of the inferior cervical ganglion) or pain in the arm from brachial plexus involvement has been noted.

Signs.—Bulging of the chest wall may occur if the tumour is parietal; mediastinal tumours may show no physical signs.

RADIOLOGICAL APPEARANCES.—The shadow is usually rounded, well defined, and in the posterior mediastinum, occasionally on the posterior chest wall. Antero-posterior, oblique, and lateral films must be taken, and Potter Bucky films of the spine.

Hour-glass Tumours of the Spinal Canal are of special interest. These grow from the posterior roots, and have a spinal portion and a larger external portion communicating through the intervertebral foramen by a narrow neck (Fig. 240). The importance of recognising them radiologically is obvious, since they are amenable to surgery. They are extrapleural and situated paravertebrally

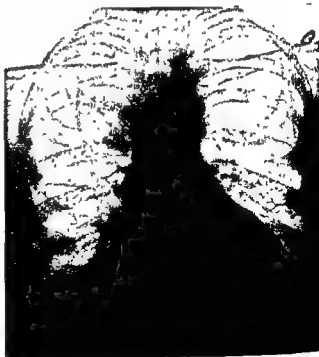


FIG. 241 — Mediastinal neurofibroma (hour glass tumour)



Lateral view. Erosion of posterior surface of bodies of fourth and fifth vertebrae, with anterior location of bodies.

FIG. 242.—Neurofibroma, dumb-bell or hour-glass type. Note erosion of posterior ends of fourth and fifth ribs and articular processes. Multiple subcutaneous neurofibromata were present, with pigmentation of the skin.

or intercostally. The reflection of the pleura over the tumour may be visible in tangential views. Symptoms and signs of spinal compression are present if the intraspinal portion is large. Artificial pneumothorax will distinguish them from pulmonary tumours.

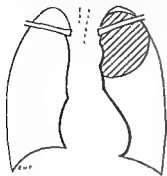


FIG 243 —Diagram
Neurofibroma (After Brown
and McCarthy)

RADIOGRAPHICALLY their appearance is very characteristic when their situation is realised. The writer has seen several cases showing a tumour, the size of a tangerine orange, projecting to one side of the spine, sharply defined, and has noted a widening of the intercostal space corresponding with the site of the tumour with slight elevation of the rib above it, apparently due to rotation of the rib: possibly it results from mechanical factors, possibly from an isolated paresis of the intercostal nerve belonging to that segment, and relaxation of the intercostal muscles, allowing upward rotation of the rib at the posterior articulation. Localised erosion of the vertebral

end of the rib and transverse process and enlargement of the intervertebral foramen and narrowing of the pedicles also occur. The rate of growth is very slow.

Multiple Neurofibromatosis.—Kienböck and Mayer report a case of multiple neurofibromatosis in a young man who had pain in the back, weakness, and wasting for some years. X-ray examination showed large nodular tumour masses in the posterior mediastinum, retro-pleural, extending in front of and alongside the vertebræ for the entire dorsal region. There were present scoliosis and multiple erosions of the lateral aspects of the vertebral bodies, heads of the ribs, and many articular processes. The diagnosis lay between lymphogranulomatosis and multiple neurofibromatosis. There were no changes in the skin or subcutaneous tissue, no palpable nodules or pigmentation.

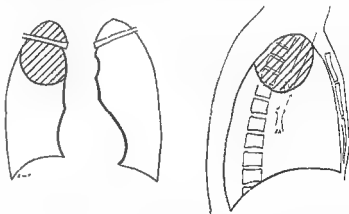


FIG 244 —Diagram: Neurofibroma (After Classen, *Acta Radiologica*, 16, 5, 1935.)

A similar case was published by Gorlitzer; subcutaneous neurofibromatosis was present in this case.

Neuroblastoma.—Neuroblastoma (neurocytoma) arising from the sympathetic is found most commonly in infants and young children. It may originate in any part of the sympathetic system. It is highly malignant. Two cases in children of 5 years, each presenting a rounded shadow at the apex, due to primary sympathetic neuroblastoma, are described by *Hartung* and *Rubert*. Metastases were present subperiosteally in the long bones and skull. Most cases are not of the hour-glass type, and produce no bony changes in the spine.

THE DIFFERENTIAL DIAGNOSIS is from malignant tumour of the ribs or pleura, sarcoma, endothelioma, or secondary tumour. These are likely to produce *invasive* erosion in the bone. A very large substernal thyroid may simulate a neurofibroma (*Chaoul*) see Fig. 236. The writer has seen a *hydatid cyst* at the back of the chest with similar radiological appearances. The cyst was flattened by contact with the chest wall, and was hemispherical in the lateral view. A *dermoid cyst* near the chest wall may also present a similar picture.

Artificial pneumothorax is necessary to exclude an intrapulmonary lesion. Encysted collections of fluid on the chest wall sometimes produce rounded shadows in the antero-posterior view, but they are not so well defined, are usually oval in shape, and in tangential views can usually be distinguished by their flattened half-moon shape and by their tapering off into thickened pleura at their edge. In case of doubt, a paracentesis should be employed.

Aneurysm of the arch has to be excluded in some of the upper mediastinal cases. Absence of pulsation, absence of cardiac enlargement or of generalised aortic enlargement, and the demonstration of an intact aortic arch by fluoroscopy must be relied upon to make the differentiation.

Tracheobronchial Cysts.—These cysts of the mediastinum do not contain tissues derived from skin, but are lined with ciliated epithelium. They have a developmental origin from the foregut, before it is differentiated into oesophagus and trachea. A tracheobronchial cyst lies in contact with the bifurcation of the trachea, and may reach a considerable size.

DERMOID CYSTS AND TERATOMA OF THE MEDIASTINUM

These are tumours of congenital origin. They are rare. In 1929 *Robertson* and *Brown* found records of 123 cases. *Heuer* 134 cases, adding four. *Moir* traced six recent cases and added two of his own, with radiographic findings. *Hedblom*, in a comprehensive review of 185 cases, discusses the ætiology and classification of these tumours. Those which contain ectodermal derivatives only he classifies as epidermoids; those which also contain mesodermal derivatives, as dermoids: those containing derivatives from all three germinal layers, as teratomata. Most are cystic, but some are solid. The whole group is conveniently included under the term "dermoids."



FIG. 243.—Dermoid cyst. M 57. A large, well defined mass, with a layer of calcification on the surface, occupies the left anterior chest, arising from the mediastinum. History: Bronchitis for 6 months, with some pain in the left upper chest. Operation: Cyst removed. It was adherent to innominate vein and pulmonary artery.

Pathology

DERMOID CYSTS.—These are cystic tumours, the walls of which have the structure of skin; the contents are hair, oily or sebaceous material, cholesterol, and occasionally teeth. Rarely the wall may calcify (Fig. 245). They arise from epithelial rests, in developmental grooves and fissures. They are always benign.

TERATOMATA.—The origin of these is disputed. They are regarded by various authorities as (a) representing an included thoracopagus twin; (b) arising from undifferentiated cells related to the ovum—a parthenogenetically developing egg (*Wilms*); (c) originating from isolated blastomeres in the early stage of development (*Budde*). These undergo limited development in that part of the embryo where they happen to lie, and may give rise to tumours in the testis or mediastinum. The teratomata sometimes contain chorionepithelioma tissue (*Hammarshjöld*) and are capable of metastasising to lung and brain. Sarcomatous or carcinomatous degeneration may also occur in 10 per cent. of the cases (*Lambert*).

Clinical Features

Dermoids and teratomata occur with nearly equal frequency in the two sexes (*Hedblom*) and are usually discovered at or before the age of 20.

Symptoms usually occur before the age of 30, and may result from increase in the contents, distension of the cyst, infection or malignant degeneration of a teratoma. The tumours sometimes enlarge rapidly at puberty. The contents of a dermoid cyst (hair, oily or sebaceous matter) may be expectorated after rupture into a bronchus—with complete cure in rare instances. Subjective symptoms are usually those of pressure, such as pain in the chest or down the arm, a sense of pressure or "congestion" with irritating cough, dyspnoea, or dysphagia. Hæmoptysis (blood-streaked sputum) is often present. Engorgement of the veins of the neck or oedema of the face may occur. The tumour may perforate into pleura or bronchi, and be encountered during operation for empyema.

A localised swelling of the ribs, sternum, or suprasternal region and localised dullness to percussion may be noted if the tumour arises, as it usually does, in the anterior mediastinum. In a case published by *Fuller and Jagger*, of a dermoid cyst at the back of the chest, at the level of the tenth and twelfth ribs, an external swelling was present beneath the erector spinæ, as well as an intrathoracic parietal shadow containing a tooth. Aspiration of cyst contents may reveal sebum, hair, or cholesterol, and assist in the differential diagnosis from hydatid cyst.

Radiological Appearances

THE DERMOID CYST shows a smoothly rounded, clearly defined, circular or oval shadow projecting from the mediastinum, usually the anterior,

sionally the posterior. Sometimes the shadow is more pyramidal. The medial aspect blends with the mediastinal shadow, or may be connected with it by a short stalk (*Kastle*), best shown in an oblique view. A tooth may occasionally be seen within the shadow (*Jagger*). Fragments of bone have also

been observed. After rupture into a bronchus air and fluid may be seen in it.

The shadow of these cysts is variable in position. Usually it is below the level of the aortic arch, at mid-level, but in cases published by *Liebmann* and by *Chaoul* it occupied the superior mediastinum, obscuring the aortic knob. In such a situation aortic aneurysm, substernal thyroid, a persistent thymus, or thymus tumour are simulated. In a case published by *Moir* it was somewhat triangular and lay in the anterior half of the chest, extending from the second to the fourth interspaces.

Dural classified them, according to position, into four groups: (1) Retro-

sternal, between mediastinal pleura and the sternum; (2) cervico-retrosternal, presenting in the sternal notch; (3) mediastinothoracic, extending into either thoracic cavity, and (4) lateral thoracic, lying largely in either thoracic cavity. The base of the tumour in the first two groups is above the middle of the sternum. The mediastinothoracic and lateral may occupy any part of the anterior thoracic cavity. In three cases of the 185 reviewed by *Hedblom* the tumour was posterior. In exceptional cases the tumour is entirely lateral, and its shadow distinct from the heart and mediastinum.



FIG. 246.—Dermoid cyst, lateral view. The large cyst, containing bony structures, displaces the sternum forward and the heart downward.



FIG. 247.—Dermoid cyst; operative confirmation.
 Removed.
 Good recovery.

Same case after lipiodol injection, showing bronchi crowded backwards by the cyst, which lay anteriorly.

Fig. 247 relates to a child of 7. During the routine examination of the chest following an attack of measles a large mass was found in the left anterior mediastinum. It was roughly oval and contained two small faint particles of bony density. A diagnosis of dermoid cyst was suggested on this evidence. Lipiodol injection showed that it was extrapulmonary, as the lipiodol-filled bronchi were crowded to one side by it. Operation showed a dermoid cyst, which was removed. It contained fragments of teeth.

Occasionally the tumour extends downwards, and appears to rest upon the diaphragm. It may even arise from the diaphragm (*Hedblom*). It may be small, about the size of an orange, or extend outwards as far as the chest wall, as occurred in one of the three cases described by *Tudor Edwards*, in which the cyst was intrapulmonary, almost completely filling one hemithorax, and covered only by a thin layer of lung tissue. It may also grow in an interlobar fissure.

The rate of growth is usually very slow. *LeWald* has observed three cases, one in a man of 40, for four years, with no change in size, one in a man of 37, for one year, with no change, and one in a young man of 19, for three years, with steady increase during thirty-eight months (from 11.2 cm. to 14.5 cm. in diameter).

An unusual radiological sign was present in the first of *LeWald's* cases. The large globular shadow in the posterior mediastinum fitted into a bed in the anterior surface of four vertebrae, which were deformed in a symmetrical curve. There was no localised erosion of the body of each vertebra, as occurs in aneurysm, but the vertebral column appeared to have been gradually curved to form a bed for the slowly developing cyst.

Pemister and others found, in a proved case of mediastinal dermoid cyst, a fluid-level within the shadow which shifted with change of position of the patient. This appearance was due, not to air and fluid, but to a separation of the fatty contents from the aqueous contents of the cyst. The fat, fluid at body temperature, floated upon the surface of the other contents; and, being more translucent, gave a shadow like that of an air bubble. The appearance was correctly interpreted before operation, and later confirmed by X-ray study of the excised cyst and of its contents. The shadow of a hair-ball could be made out in these later studies just protruding above the level of the aqueous contents. The contrast between fat and other contents was insufficient to allow the fluid-level to be seen on the screen.

Teratomata, as described in the literature by most observers, show rounded, smoothly contoured shadows undistinguishable from dermoid cysts, always situated in the anterior mediastinum. *Hammarskjöld*, however, describes in his two cases a lobulated or wavy contour, due to the different types of tissue in the wall, and believes that this evidence of irregular structure distinguishes them from the cystic dermoid tumours. In all other respects the two conditions are radiologically exactly similar. Chorionic tissue is present

in about 20 per cent. In these cases concomitant testicular atrophy or even gynæcomastia may be present. The Asheim Zondek test may be positive.

The Differential Diagnosis is from encysted empyema, aortic aneurysm, hydatid cyst, substernal thyroid, thymus tumour, paravertebral abscess, bronchogenic carcinoma, mediastinal tumour, lymphosarcoma, lymphadenoma, or other enlargements of the lymphatic nodes, fibroma and lipoma.

LIPOMA OF THE MEDIASTINUM

These rare benign tumours resemble dermoids and teratomata, in that they occur in the anterior mediastinum and project into the lung fields with well-defined contours. Sometimes they are unusually fatty teratomata, and their true nature is then only determined by rigorous histological investigation of all parts of the specimen.

Lipomata are sometimes partly intra- and partly extrathoracic, communicating by an isthmus through the sternum or elsewhere, or extending upwards into the neck. Though encapsulated, they are sometimes so large that surgical removal is a matter of great difficulty. A case of mediastinal lipoma has been reported by *Schinz* and *Gasser*.

FIBROMA

Twenty cases of this tumour have been recorded. They may lie in the anterior mediastinum, or may occur elsewhere on the chest wall. They show appearances in the X-ray indistinguishable from other benign tumours. They are, as a rule, circular in outline, but tangential views may show a broad base of origin from the chest wall. Mixed forms may occur, e.g., Fibroleiomyoma (*Jacobaeus* and *Einar Key*).

A case of thoracic fibroma in a female patient aged 37 was published by *Iles*. The rounded shadow lay in the lower thorax. It was not stated whether the tumour was of neurogenic origin.

RARE CONDITIONS SIMULATING MEDIASTINAL TUMOUR

In the diagnosis of mediastinal tumour the following rare conditions must be considered: innoctinate aneurysm, cardiac aneurysm, neoplasm of the pericardium, pericardial diverticulum, mediastinal effusion, interlobar effusion at the upper end of the main interlobe near the mediastinal surface of the lung, hydatid cyst near the mediastinum, and gumma.

Diverticulum of the Pericardium.—This is a rare tumour, described by *Kienbock* and *Weiss*. It shows a projection from the cardiac shadow which is rounded, sharply defined, and pulsating. It usually occurs on the right side; the aorta is not enlarged. It results from chronic adhesive pericarditis. One of the three patients whom these authors first saw in 1929 has recently been re-examined. He shows the appearances which they consider typical. A shadow with a convex, slightly lobulated contour projects from the right heart.

border. A fine line of calcification outlining the inner wall of the serous pericardium is bordered by a thicker outer non-calcified shadow of the fibrous layer; in the lateral view the diverticulum lies in contact with the anterior chest wall.



FIG. 248.—Neurofibroma F 40 History 3½ years' pain and weakness in right arm and hand. Miosis right eye. Trachea and œsophagus displaced. Operation histological structure fibroma.

Morris, in this country, has published a case having rather similar appearances. The fine line of calcium was clearly visible in the radiogram. In the lateral view the shadow lay over the origin of the ascending aorta. It seems probable that this case is also a pericardial diverticulum. A pericardial origin was considered possible by *Morris*. *Jansson* described a case of pericardial diverticulum in a girl of 16, and observed that during inspiration the shadow became long and narrow, but widened again on expiration. This "moulding" with respiration proved the swelling to be of a soft or fluid consistency, and was a point in the differential diagnosis against tumour. The diverticulum consisted of two pockets, one of which, having a freer communication with the pericardial sac, pulsed more

freely than the other. There was no trace of calcification in the wall.

Other conditions which are not rare but which may be mistaken for mediastinal tumour are: (1) Scoliosis; (2) Paravertebral abscess in Pott's disease of the spine; and (3) A dilated œsophagus in high-grade cardiospasm. The differential diagnosis is easy with care.

MEDIASTINITIS

Mediastinitis may be acute or chronic. Abscess may occur from perforation of the œsophagus, suppurative lymphadenitis of the mediastinal glands, or following trauma. A subacute mediastinitis occurs in pericarditis, pleurisy, pneumonia, and syphilis. An abscess cavity with fluid-level may be seen in mediastinitis; and on one occasion the writer saw barium enter a ragged cavity in the mediastinal tissues, in a case of perforation of an œsophageal carcinoma. Chronic indurative mediastinitis with adherent pericardium may occur; the

peritoneum may be involved, with perihepatitis, cirrhosis of the liver, and ascites (Pick's disease).

Radiological Appearances.—In mediastinitis the mediastinal shadow is increased in breadth and in density, particularly in the upper half. The contours of the median shadow are well defined, and run more or less vertically, sometimes showing a slight convexity outward (*Assmann*). The strongly convex or lobulated contour typical of neoplasm is not seen. In the lateral view the retro-sternal and posterior mediastinal clear spaces may be opaque.

In chronic mediastinitis, in addition to the broadening and increased density of the median shadow, various changes may be observed. due to the inflammatory changes in the pericardium and pleura. Adhesions between pericardium and pleura. loss of definition of the cardiac outline. pulmonary congestive changes, pleural effusion on one or both sides, pleural thickening, a filling up of the cardio-phrenic angles, and a limitation of movement of the diaphragm may be evident. What movement there is may leave the heart "suspended" in the chest as a result of mediastinal rigidity and shortening (*Morton*). If there are marked pericardial adhesions, an upward movement of the left half of the diaphragm with each cardiac systole, or a similar inward movement of the lower posterior ribs, has been observed. Adhesion between the trachea and aorta may lead to the unusual finding of pronounced upward movement of the aorta on swallowing.



FIG. 249.—Mediastinal abscess following perforation of œsophagus. Carcinoma. Barium has entered the ragged abscess cavity

HÆMATOMA OF THE MEDIASTINUM

A mediastinal hæmatoma may result from injury to a large vessel in the root of the neck; the blood gravitates downwards into the mediastinal areolar tissue. The mediastinum is widened and shows straight vertical borders, as described under mediastinitis (*Lenk*). If the hæmatoma occurs from rupture of the aorta or aortic aneurysm (dissecting aneurysm), a more localised widening is present. Rupture into the pleural cavity is common. If due to active hæmorrhage, rapid change in size of the shadow is to be expected.

CHAPTER XXXI

PRIMARY NEOPLASMS OF THE LUNG

CANCER of the lung would appear, from a consideration of post-mortem statistics, to be on the increase. It is, however, disputed whether this increase is real or apparent. The statistics are reviewed by *Davidson* in his book, *Cancer of the Lung*, and the possible fallacies in the statistical evidence are also discussed by *Hadfield* and *Garrod*.

Ætiology.—No satisfactory explanation is forthcoming for the apparent increase in the incidence of pulmonary cancer. Influenza has been arraigned as a possible ætiological factor, and attempts have been made to correlate an increase in lung cancer with recent epidemics of influenza, against this view the entire absence of lung cancer in Iceland, which has suffered severely from influenza appears to be a conclusive argument. Tuberculosis does not predispose, but coexists in about 7 per cent. of the cases (*Davidson*). Pneumoconiosis ordinarily does not predispose to lung cancer, but there is one pure example of an occupational type, namely that which occurs among cobalt and bismuth miners of Schneeberg in Saxony, studied by *Rostoski*, *Saupe*, and *Schmorl*.

Incidence.—Primary lung cancer is stated to be four times as common in men as in women, and usually occurs between the ages of 40 and 60. The writer's experience is that it is fifteen times as common in men as in women, and its extreme rarity in the female sex should render the radiologist very wary of making this diagnosis, unless the findings are quite typical, in a female patient.

Sarcoma is much less common. Primary chondrosarcoma and osteosarcoma may occur in the lung, but are very rare.

Morbid Anatomy and Histology.—Up to recent years two histological types of the disease were described: bronchial carcinomata and "lymphosarcoma" or "sarcoma" of the mediastinum. The latter, being composed of small round or oat-shaped cells, was at one time supposed to be sarcomatous, but is now accepted, as a result of the work of *Turnbull*, *Barnard*, *Dagnid*, and *Shewman*, as true carcinoma.

Radiological literature in past years reflected this fallacy, and most of the illustrated cases of infiltrating tumours extending outwards from the mediastinum, described in the older literature as lymphosarcomata, would nowadays be correctly described as carcinomata. True mediastinal lymphosarcoma does, of course, exist, but is comparatively rare.

Carcinoma of the lung may therefore be reclassified histologically as follows:

(1) SQUAMOUS CARCINOMA.—Composed of large cells, and supposed to arise in metaplastic bronchial epithelium, it forms a hard white growth in the lung root. Metastases are small and discrete, and are usually confined to neigh-



←Primary growth
in bronchus.

FIG. 250.—Specimen : Small carcinoma of bronchus with metastasis to the glands of the tracheal bifurcation.

bouring lymph nodes. The average age of incidence is 46.6 years. *Ewing* has described a type of carcinoma arising in the alveoli, the epithelium of which is squamous. *Karsner* and *Saphir* state that there is no evidence that cancer of the lung originates elsewhere than in the bronchi.

(2) **COLUMNAR AND SPHEROIDAL-CELLED CARCINOMA.**—This may arise in the bronchial epithelium or mucous glands. The cells are large, columnar, or cuboidal, sometimes mucus-secreting and having a tubular or alveolar arrangement. The less differentiated forms are bulkier, softer, and more widely disseminated. The mediastinal glandular masses are frequently confluent. They metastasise freely. *Moise* (quoted by *Karsner*) gives the following order of frequency of such metastases: regional lymph nodes, liver, kidney, lung,



FIG. 231.—Specimen carcinoma of bronchus: the tumour is nearly blocked by a nodular tumour, which has perforated the wall. From this point the disease extends through the lung towards the apex.

pericardium, abdominal glands, pleura, brain, adrenals, bone, cervical glands, and heart. The average age of incidence is 51 years.

(3) **ROUND-CELLED AND OAT-CELLED TUMOURS.**—The cells are smaller than in the preceding, with large nuclei; unlike sarcomata, the tumours have well-formed blood vessels. Macroscopically they resemble the columnar type, but may be even larger, with a great tendency to degeneration. They are highly malignant and have a lower age incidence (average age 43 years). They

metastasise freely, to the same situations as the columnar-celled. Very little distinction can be made in practice between the different types. Radiologically it is hardly possible to distinguish one from another. *Ewing* and *Sante* have described an "alveolar" type, highly malignant, which rapidly converts the lung into a solid mass.

Origin and Spread.—The growth usually starts in the wall of a large bronchus, not far from the point of division of the main bronchi, and spreads to the neighbouring hilar and mediastinal glands. Nodular growths projecting into the lumen tend to produce early bronchial stenosis. Flat growths tend to spread in the submucosa without alteration. After perforation of the bronchial wall, the growth may spread rapidly along the peribronchial lymphatics, in part accounting for the hairy edge frequently seen in radiograms. The bronchi are surrounded by cuffs of growth. Nodules may be found upon these radiating strands, or on the pleura.

In a few instances the growth originates in a smaller bronchus, well away from the hilum, and forms a rounded nodule or wedge-shaped infiltration. The glands of the hilum are usually affected late and may be little in evidence in a radiogram. A band of infiltration usually, but not invariably, extends from such a growth to the hilum, which can be recognised in the radiogram. The right lung is affected with greater frequency than the left, and the right upper lobe a little more frequently than the others. *Kikuth* (cit. *Davidson*) gives the following distributions :

Right upper and middle	38
Right lower	35
Left upper	31
Left lower	30

Distant Metastases.—These occur in many organs. Metastasis to the brain is frequent, and such cases have been operated upon in the belief that a primary brain tumour is present. The skeleton, liver, adrenals, kidneys, pancreas, spleen, and ovary may also be affected.

Symptomatology.—The onset is usually insidious. Many cases have been found by chance by radiological examination, often in the course of an opaque-meal examination, the leading symptom being loss of weight and dyspepsia. The majority of cases, however, have symptoms definitely related to the respiratory tract. In the most insidious type the disease is for a long time regarded as a chronic bronchitis. Chronic tuberculosis in elderly people may often masquerade as neoplasm, or *vice versa*. The writer has seen several cases of bronchial carcinoma which dated their illness from an attack of influenza, either as a result of coincidence, or because of a sudden exacerbation of symptoms which had passed almost unnoticed up to the time of the influenzal attack. These cases are particularly difficult, for the radiological appearances of an infiltrating neoplasm and of an inflammatory pneumonitis may be identical, judged on a single examination.

The disease may be ushered in more abruptly by hæmoptysis, or by pleurisy, dry or with effusion, the latter usually bloodstained.

Cough, with scanty expectoration at first, is almost invariably present. If the recurrent laryngeal nerve is involved, the cough is brassy and the voice

hoarse. In some cases the presence of malignant cells in the sputum confirms the diagnosis. *Hæmoptysis* is usually frequent and small. When bronchiectasis or necrosis occurs the sputum is purulent or foul. *Pain* is variable, and usually due to involvement of the pleura or ribs. It is sometimes of the "cardiac" type. Apical neoplasms may produce pain in the arm, with wasting, due to involvement of the brachial plexus. *Dyspnœa*, *weakness*, and *loss of weight* are perhaps the most frequent symptoms, but dyspnœa is often slight in the case of a nodular growth in the parenchyma unaccompanied by bronchostenosis or marked enlargement of the mediastinal glands. *Paroxysmal tachycardia* has been recorded (Leichtner) as a result of involvement of the sympathetic and vagus by mediastinal glands. *Cyanosis* is usually only present in advanced cases, unless the superior vena cava is obstructed, a complication which may occur suddenly or gradually. *Clubbing of the fingers* is usually slight or absent. *Pulmonary osteoarthropathy* may occur in cancer of the lung, but is rare owing to the short course of the disease.



FIG. 252.—(Lime hypertrophic pulmonary osteoarthropathy in the same case as shown in Fig. 253

The duration in the majority of cases is from six months to one year. The younger the patient, the more malignant the disease tends to be. In rare cases the duration may be two years or over.

RADIOLOGICAL FEATURES

The radiological appearances are protean. It is impossible to give any satisfactory classification of the radiological types, though such classifications have been attempted.

The difficulties encountered in making such classifications have usually forced the observer to limit himself to a few primary types. Thus the classifications of Otten, Asmann, Schinz, and recent American writers are, essentially, reducible to two main forms :

- (1) Hilar carcinoma.
- (2) Parenchymatous or lobar carcinoma.

Other writers have included in their classification what are really momentary appearances or localised anatomical forms of these two.

The appearances which we see radiologically are momentary pictures, and serial examinations at short intervals of time often show a rapid change from one "type" to another. To take simple examples, a hilar "type" may rapidly become converted, with the abrupt onset of atelectasis, into a lobar form; a nodular parenchymatous carcinoma may change in a few weeks or months into either a "cavernous" or diffuse type. In similar periods an atelectatic "type" may change to a bronchiectatic, or develop an effusion, or spread to the opposite lung in diffuse or nodular form. A neoplasm originally confined to one lobe, and therefore classified as lobar, may spread to other lobes, or become masked by effusion, or even spontaneous pneumothorax.

Bronchostenosis may in some cases clear up spontaneously, or, more frequently, be induced to clear up by radiotherapy. A study of serial radiograms of many cases will eventually lead to a better understanding of what has already happened, is happening, and is likely to happen in a given case of bronchial carcinoma than any classification, and is essential if we are to understand the "momentary" picture. In any doubtful case, if the patient has been X-rayed by another radiologist or hospital, it is very desirable to obtain the previous film for comparison: much help may be obtained from it, especially in the differentiation of cavernous growth from abscess.

It is unfortunate that most cases do not come to post-mortem, and that a correlation of radiological and pathological findings is so often denied us. If we do see a post-mortem, it may fail to explain the X-ray appearances which have puzzled us, owing to progression of the lesion in the interim.

The radiological appearances depend upon many factors: (1) site of origin; (2) direction, rate, and method of spread; (3) complications—bronchial occlusion, bronchiectasis, infection, pleural involvement, necrosis, cavitation, and (4) effects of treatment, e.g. paracentesis, bronchoscopy, operative removal of the primary tumour, radiotherapy, pneumothorax.

The site of origin may be central, in a large bronchus, or peripheral

- (a) In a branch bronchus, or
- (b) According to *Ewing*, in alveolar epithelium.

In the central type massive glandular enlargement and early atelectasis of an entire lobe are most likely to occur. A great majority of carcinomata arise near the first division of the main bronchi and quickly invade the hilar glands, so that hilar enlargement is an early sign. If the patient presents himself at this stage, we see a radiographic picture of the hilar type (Fig. 253).

In the less common peripheral type, hilar enlargement is usually later and less massive. The peripheral type, in its early stages, may take one of two forms:

- (a) If a medium-sized branch bronchus is the site of origin, a wedge-shaped area of shadow, with its apex towards the hilum, is seen. It corresponds in situation to the bronchopulmonary segment involved, and resembles a pne

monic consolidation. It is due partly to growth, partly to atelectasis peripheral to the growth limited to the territory supplied by the bronchus concerned.

(b) If the growth arises in a small bronchus, it soon penetrates the wall and grows as a rounded nodule in the lung tissue. This gives rise to the less common nodular or lobar type. Atelectasis is not a marked feature, since the bronchus of origin is small. The isolated nodule grows steadily in size, and may retain



FIG. 273 —Bronchial carcinoma of "hilar type." Bronchoscopy showed primary in left bronchus.

its rounded form until very large, or it may at any stage of growth infiltrate the surrounding lung, and so show a blurred and fuzzy edge in the radiogram. In some cases the continued growth of the tumour converts the whole lobe into a solid mass, and bulges the interlobar pleura towards the contiguous lobe or lobes.

Although the above subdivision into central ("hilar") and peripheral parenchymatous types corresponds in the main with the observed facts, it must not be too rigidly applied. Not every carcinoma of a large bronchus gives rise to a massive hilar tumour, nor does it often remain restricted to the bronchial lumen. Like the peripheral forms, it may penetrate the bronchial wall early,



FIG. 254.—Bronchial carcinoma in apex of right lower lobe, simulating hilar carcinoma.

Lateral view. The carcinoma, of peripheral origin, occupies part of the apical segment of the lower lobe.

and grow as a circumscribed or diffuse tumour in the lung. A cancer of a bronchus and its glandular metastases, like a cancer of the breast and its glandular metastases, obeys no strict laws, as to relative size of primary and glands, as to date of appearance of the metastasis, or as to distribution and spread of the primary. Lung cancer appears, indeed, to be even more lawless



FIG. 255.—Carcinoma of right upper lobe. Note the intact interlobar septum which is bulged downwards by the bulky growth, but acts as a barrier to its spread. M. 32. Onset of symptoms dated from attack of influenza six months previously.

in its manner of development and rate of spread, since it finds in the lung abundant choice of pathways, and is less vigorously opposed by normal tissue

ANALYSIS OF X-RAY APPEARANCES OF PRIMARY CARCINOMA OF THE BRONCHI

There are four primary elements to be considered in attempting to analyse the radiographic picture of the common types of bronchial carcinoma.

- (1) The endobronchial tumour.
- (2) The extension of the growth into the lung.
- (3) Atelectasis in the region supplied by the bronchus.
- (4) The hilar glands.

(1) The Endobronchial Tumour

For descriptive purposes we may consider this as an endobronchial carcinoma arising from the mucosa, usually from a large bronchus close to the hilum, occasionally from a medium-sized bronchus farther out in the lung. The bronchus may, of course, have been invaded from outside by a secondary deposit in the neighbouring glands, or, as in a case reported by *Ross Golden*, by an endothelioma of the pleura, arising in the vicinity. A study of sixty cases of secondary carcinoma of the lung by the writer has produced several examples in which this has apparently occurred. In these cases the description given below would apply—that is to say, secondary carcinoma may occasionally produce X-ray appearances indistinguishable from primary carcinoma of the lung.

The endobronchial tumour cannot be directly seen in a plain X-ray film. Endoscopy is often required, and it is a function of the X-ray to indicate as nearly as possible where the growth is, for the assistance of the surgeon. Its approximate position can usually be inferred from the X-ray appearances. It may be directly demonstrated by lipiodol injection which will show (1) narrowing or irregularity of the bronchial lumen; (2) obstruction of the lumen with a rat-tailed tapering at the site of the growth (Figs. 256, 257).

The most exact and critical definition of the bronchial stenosis is obtained by the serial method of *Fariñas*. The desired bronchus is filled through a nasal catheter inserted under fluoroscopic control, and serial radiograms, controlled by fluoroscopy, are made in the lying position during filling. This method appears to have very considerable advantages over that usually adopted.

Tomography may be tried, as an alternative or as a preliminary to lipiodol injection or bronchoscopy. The writer has found that this method will show the bronchial narrowing and obstruction quite clearly in many cases. The tomogram must be taken in the plane of the bronchus; and shows the

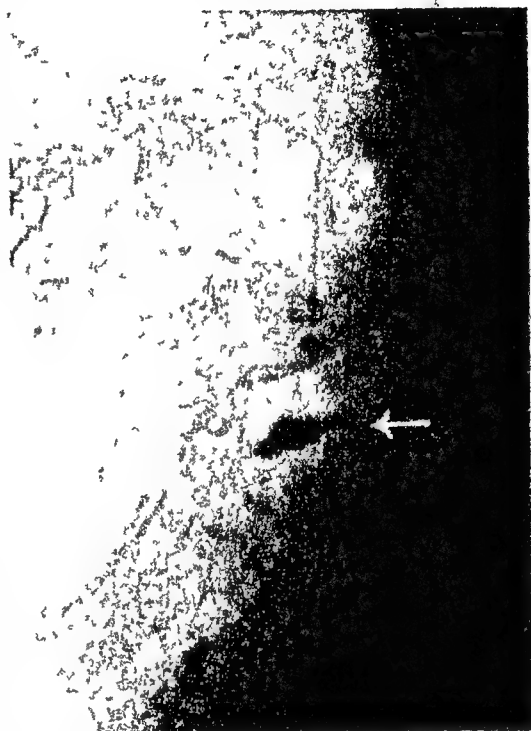


FIG 256 —Bronchial carcinoma right lower lobe. Lipiodol shows typical narrowing and obstruction ("rat-tailing") of the affected bronchus.



Fig. 27 - Bronchial carcinoma. Lateral view shows obstruction of left bronchus, with bronchiectasis at apex (sides reversed)

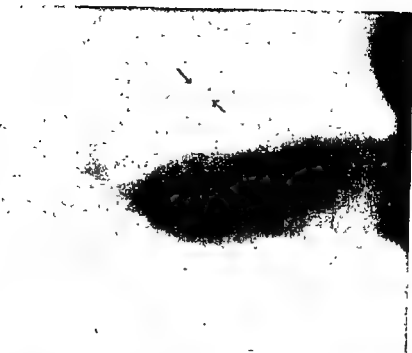


Fig. 28 - Bronchial carcinoma. Tomogram, showing stenosis of the left bronchus (arrows).

tion of the trachea, the main bronchi of both sides and their branches. The method succeeds in picking out the bronchus even though the surrounding lung is densely consolidated, or surrounded by effusion.

(2) The Extension of the Growth into the Lung

This may be local or diffuse. It may spread locally through the lung parenchyma, using the stroma and alveolar walls as a preformed framework, and gradually obliterate and replace the alveoli. Or it may grow out along the peribronchial lymphatics in the form of cuffs of peribronchial growth (Fig. 259), or spread more diffusely by retrograde permeation of the lymphatics of the lung or pleura.

The local form appears to be the more common. Post-mortem examination has repeatedly shown that the actual growth does not extend very far out into the lung, but involves only a limited region near the hilum, quite incommensurate with the large shadow shown in the X-ray. This is often the case, even when the X-ray shows a total opacity of a whole lobe. The greater part of the opacity is then not due directly to the growth, but to other factors.

Local extension sometimes follows an interlobar fissure, sometimes surrounds and involves all the branches of the lobar bronchus, and in not a few cases is limited to one or two divisions of the lobar bronchus, in such a way



FIG. 259.—Specimen: Bronchial carcinoma. The bronchial walls are infiltrated with growth and the lumen narrowed. Note lymphatic extension of the growth, in the form of peribronchial cuffs, into lower lobe is well shown.

the actual tumour and the secondary changes occupy only a segment of the lobe. These segmental forms often simulate tuberculosis or other inflammatory disease, and will be referred to later.

The local extension of the tumour is shown as a shadow, which lies close to the hilum and blends with the mediastinum. This is the picture presented by

more than 50 per cent. of all cases when first seen. It may be :

- (1) Circumscribed, or
- (2) Diffuse.

(1) *The circumscribed shadow* projects from the hilum with a well-defined border, and may be semi-lunar rounded, hook-shaped, oval, or even reniform in shape. It is uniformly opaque. The vascular shadows are lost in it, but the translucent bronchi can be traced through it. In part the tumour tissue may be derived from carcinomatous, tracheobronchial, and bronchial glands, but it does not necessarily represent a glandular enlargement. In any case, tumour tissue soon breaks through the capsule of the glands, and the shadow represents an actual nodule slowly invading the lung.

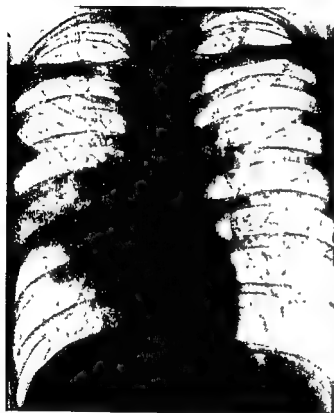


FIG. 260—Bronchial carcinoma, hilar type with diffuse peribronchial lymphatic spread into lung

(2) *The diffuse shadow* is of variable extent, usually spreading fanwise into a lobe; its outer margin has a blurred hairy appearance, due in part to peribronchial extension of the growth, in part to the emergent arterial and venous shadows, it fades off towards the periphery (Fig. 260). Sometimes it forms a dense triangular shadow close to the hilum, sometimes it is less dense and more diffuse. The circumscribed nodule may at any time change into the second form, with a diffuse spreading edge. Otten (cit. Assmann) has drawn attention to a diffuse form of primary bronchial carcinoma, resembling the diffuse form of

secondary carcinoma. The writer has seen several cases of this type. The distinction from an inflammatory lesion is extremely difficult. Clinically dyspnoea is a prominent feature. The radiogram may show no hilar mass, but merely a shadow composed of radiating strands extending outward from the hilum. This form seems to be more common in the lower lobes.

If the case is first seen in any of the stages just described, it constitutes the "hilar type" of most writers. It is usually only a transition stage. The writer has never seen such a tumour spread outwards to involve a whole lobe by direct extension. In nearly every case atelectasis supervenes before the hilar tumour reaches the size of a fist.

(3) Atelectasis in Bronchial Carcinoma

This may occur in one of two ways :

(1) By blocking of the lumen by the endobronchial growth and the surrounding mucosal inflammation.

(2) By gradual compression from without.

In either case it may come on gradually, but more often in the writer's experience it has supervened rather suddenly. *Ross Golden*, discussing the question in his paper written in 1924, says that it occurs gradually, and usually follows the development of bronchiectasis. In a case of endothelioma surrounding and constricting the upper lobe bronchi, published by *Ross Golden*, it came on gradually and involved the whole lung in two months. In a second case of a very slowly growing bronchial carcinoma of the right lower lobe, atelectasis steadily progressed over a period of fifteen months, and finally involved the whole lobe. In a third case an area of atelectasis, limited to the lower half of the right upper lobe, involved the whole lobe five weeks later. The writer's experience has usually been on the contrary. Atelectasis in most cases occurs rather suddenly, and may be extensive in a short period. In a case of right upper lobe carcinoma observed by the writer, with a hilar mass, the atelectasis, just commencing in the first film, had completely involved the whole upper lobe three weeks later. In another month, following X-ray treatment, the hilar mass had disappeared, and the atelectasis had entirely cleared. The prompt result of X-ray treatment suggests that the rather rapid atelectasis had resulted from the plugging of a bronchus by an endobronchial tumour, rather than from compression from outside. There was no evidence of bronchiectasis in this case.

In other cases a rapid onset of atelectasis and spontaneous re-aeration of the lung has been observed by the writer, without X-ray treatment. A consideration of these cases convinces the writer that if conditions are favourable, especially in the case of pure plugging by endobronchial tumour, the onset of atelectasis may be sudden, and not a consequence of infection or bronchiectasis. In the case of compression from without it may occur more gradually. The possible influence of inflammatory reaction in the mucosa, and of hæmorrhage



Fig 261.—(a) Bronchial carcinoma, F. 54. Complete opacity left lung. Atelectasis with effusion.



(b) Same case. Condition after deep X-ray therapy. Effusion cleared, upper lobe re-aerated, lower lobe still collapsed.

in the production of an atelectasis must also be considered. In tuberculous disease we know that a hæmoptysis may produce a sudden and transient atelectasis. It is quite possible for this to occur in bronchial carcinoma also.

RADIOLOGICAL SIGNS OF ATELECTASIS.—In the lung there is loss of translucency of the region supplied by the occluded bronchus. This may be a segment of a lobe, a whole lobe, or an entire lung. The opacity may be at first, and for a considerable period, subtotal, so that the ribs and some of the vascular markings are seen through it. Later the shadow becomes denser and usually somewhat smaller, and the vessel markings are lost. In the atelectatic region the opacity is homogeneous. Unless the atelectasis is very complete, there is usually a stage at which two elements can be recognised :

(a) Near the hilum, the denser shadow of the tumour and its associated glands.

(b) Extending outwards to the periphery, the shadow of the atelectatic lung. Diminution in size of affected lobe is nearly always demonstrable.

The interlobar margin of an affected lobe should be looked for in a suitable projection. It will always be found to be displaced towards the atelectatic area and is usually concave. Into the concavity is fitted the adjoining lobe or lobes, often emphysematous.

RIGHT UPPER LOBE ATELECTASIS.—If the whole lobe is involved, the postero-anterior view shows characteristic sharp delimitation by the upward arching, upwardly displaced interlobar margin. In the lateral view the line has a form resembling that shown in Fig. 149 (lateral view).

It frequently happens that the pectoral branches escape. The atelectasis then fails to reach the anterior part of the lobe or the middle fissure. The sharply defined boundary line is not seen in the postero-anterior view in such a case. The lateral view will show a triangular opacity in the apical or axillary segments, sharply delimited by the *main* fissure. Conversely the pectoral segment may be affected alone, or in combination with one of the others. The appearances of these partial or segmental atelectases are similar to those of segmental pneumonia, Figs. 152, 153.

LEFT UPPER LOBE ATELECTASIS.—If the atelectasis is very complete, the lobe may shrink into a small triangular area in the left apical region. This is, however, rare with a growth. More often the lobe is bounded by an oblique line, in the postero-anterior view, running diagonally downwards and inwards to the hilum, and slightly convex downwards. The line may be displaced upwards and inwards from its normal position. In the lateral view the interlobar margin is very clearly seen, running diagonally downwards and forwards, displaced forwards, and often concave, cf. Fig. 150 (lateral view).

In hilar carcinoma, with a well-marked hilar mass, and atelectasis of an upper lobe, an S-shaped curvature in the postero-anterior view has been pointed out by *Ross Golden* as characteristic. The inner part of the curve, convex downwards, is formed by the hilar mass. The outer part, arching upwards, is

due to the retracted interlobar surface of the affected lobe. This sign is most frequently observed in the right upper lobe, but has been noted in the left.

LOWER LOBE ATELECTASIS.—The lobe generally shrinks into a triangular form. Its outer margin may be distinct, but is often indistinct unless the patient be rotated into an oblique position. The reason for this is demonstrated in Fig. 138.

In the lateral view the main fissure is displaced downwards and backwards; the posterior *cul-de-sac* is shallow, owing to elevation of the posterior part of the diaphragm.

Segmental atelectasis may also occur in the lower lobe.

MIDDLE LOBE ATELECTASIS.—This lobe may be affected alone by a carcinoma blocking its bronchus. In the postero-anterior view it shows a triangular opacity, bounded above by the horizontal fissure, to the outer side by the main fissure, which is displaced inwards, and not as a rule very sharply defined. In the lateral view the typical middle lobe triangle is seen smaller than a normal middle lobe, and sometimes reduced to a narrow band.

The lower lobe sometimes bulges into its concave lower surface. In one of the six cases of middle lobe carcinoma observed by the writer, the growth at autopsy occupied only the proximal part of the lobe, and nowhere reached the surface. The X-ray opacity, which filled the entire lobe, was proved by the autopsy to be due in the main to atelectasis.

TOTAL ATELECTASIS.—Total atelectasis of a whole lung results in opacity of the whole hemithorax, with displacement of the heart and mediastinum to the affected side. This is a fairly common occurrence. The trachea is usually straight, but directed obliquely towards the base, but it is sometimes curved towards the affected side. This should be remembered, because it has been stated that this curvature only occurs in fibrosis and not in atelectasis. If the atelectasis clears up the trachea immediately resumes its normal position.

Displacement and curvature of the trachea to the opposite side in such cases may be due to one of two causes

(a) Effusion, in considerable quantity.

(b) Malignant tracheobronchial glands on the affected side.

If effusion is present in any quantity the mediastinum may be displaced as a whole towards the opposite side; more often the effusion and atelectasis "cancel out," so that the heart and mediastinum are not displaced at all.

EFFECTS OF ATELECTASIS ON OTHER STRUCTURES

The Chest Wall.—The chest walls fall in on the affected side, and the ribs slope more steeply ("roof-tiling"). The intercostal spaces are narrowed. This tends to affect the upper ribs only, in upper lobe lesions, and the lower ribs in lower lobe lesions. Scoliosis may occur.

Diaphragm.—The diaphragm is usually raised. It may maintain its normal bow shape, but often shows elevation of the central part in the cardio-phrenic

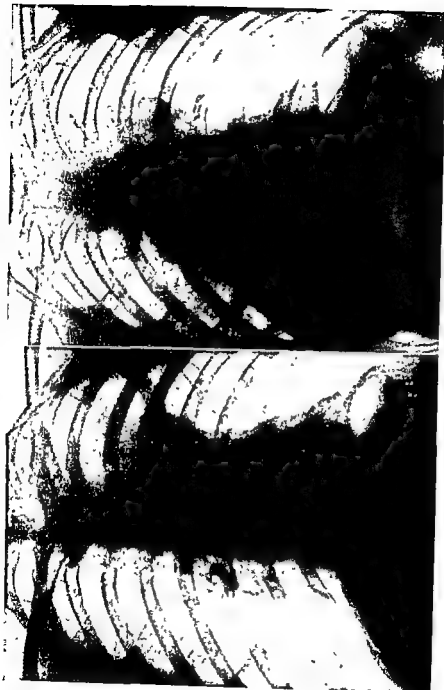
angle, which is obliterated by a continuation of the diaphragm shadow on to the lower part of the mediastinum. This peculiar shape of the diaphragm is difficult to explain. *Assmann* suggests that the central peak is due to the hepatic veins, but this is impossible, since the hepatic veins join the inferior cava in the substance of the liver, and have no extra hepatic course. In the lateral view the posterior part of the diaphragm is elevated in lower lobe atelectasis. The anterior end is often raised in middle or upper lobe atelectasis. Diaphragmatic movements are diminished. Occasionally the phrenic nerve is compressed by the growth and paralysed, leading to complete loss of downward movement of the diaphragm or to paradoxical movement. Phrenic paralysis is common in extensive lesions, and is more common on the right side.

Mediastinum.—The obstruction of air entry into the bronchus by delaying the inflation of the lung allows the other lung, which is free to inflate, to occupy more than its fair share of the total thoracic space. The mediastinum therefore moves across to the affected side in inspiration (*Holzknicht-Jakobson* sign). *Lenk* has shown that this inspiratory movement towards the affected side may be brought about by a sudden short forcible inspiration ("mediastinal jerking") in cases of partial stenosis which have shown no mediastinal movement on ordinary deep breathing. Later there may be a permanent displacement of the heart and mediastinum towards the affected side. Lateral displacement of the heart is often absent in cases of pure lower lobe atelectasis; but backward displacement can be observed in the lateral view. An inspiratory displacement of the anterior mediastinum may occur at the retrosternal "weak spot": especially if the rest of the mediastinum is rigid as a result of infiltration by growth. The opposite lung then bulges across at this site, and is seen as a translucent area bounded by a curved line; its anterior position is established by slightly rotating the patient, and noting its movement relative to the great vessels. In the lateral view the retrosternal clear space is increased in antero-posterior depth.

Valvular Obstruction.—This condition may be observed in the early stages of bronchial carcinoma, but is rare. The powerful inspiratory movement, due to the dyspnoea, sucks air into the lung, past the obstruction, which cannot be completely expelled. There results an over-distension of the lung, with displacement of the mediastinum to the sound side in expiration. The appearances, as in the case of obstruction due to foreign body in a bronchus, are brought out more clearly by observation during forced expiration; during inspiration the condition may not be obvious. (See Fig. 262.)

When one hemithorax is completely opaque and the mediastinum and heart are drawn towards that side, the following points may be of use in distinguishing between chronic indurative pneumonia (fibrosis of the lung) and massive opacity due to growth.

(a) In fibrosis the opacity is often less absolute, and the shadow of the displaced heart may be visible through the general shadow.



(a)

(b)

FIG. 202.—Mediastinal movement (Holzknecht-Jacobson sign) in bronchial obstruction by neoplasm. (a) Inspiration, mediastinum displaced towards the affected side. (b) Expiration, mediastinum displaced toward normal side, while affected side is hyperaerated (alveolar obstruction).

(b) In fibrosis a strong angulation of the trachea towards the affected side may be present. It is indicative of an old apical fibrotic lesion.

(c) In neoplasm, the trachea is displaced to the affected side as a rule, and is usually straight, but directed obliquely. Occasionally the writer has found that while the trachea is displaced as a whole towards the opaque side, there is, at and above the bifurcation level, a local bulging of the upper trachea away from the opaque side. This is due to enlarged glands in the hilum, and is the only evidence of their presence: their outer edge is lost in the shadow of the effusion.

In very rare cases there is a marked mediastinal displacement towards the *non-opaque* side due, not to effusion, but to a very bulky tumour completely filling the lung. *Sante* has described such a case, due in his opinion to a highly invasive type of carcinoma which fills the lung without regard to interlobar barriers. The writer has seen one similar case in which repeated explorations failed to withdraw any fluid, but met everywhere with solid resistant lung. In this case the marked mediastinal shift to the opposite side may have been due to this kind of tumour. (No autopsy.)

(4) The Hilar Glands in Bronchial Carcinoma

The first station for the arrest of metastases from carcinoma of a bronchus is in the lymphatic glands of the lung. Next, the hilar glands and tracheo-bronchial glands are affected in succession. From here communications lead to the anterior and posterior mediastinum. In the anterior mediastinum special importance attaches to a group of glands at the division of the superior vena cava into the two innominate veins, with which the phrenic nerve is closely related. On the left side there is a corresponding group of glands between the aortic arch and the left innominate vein, to which the left recurrent laryngeal is closely applied. In the posterior mediastinum the lymph channels along the oesophagus are important. Here there are free communications between right and left sides. *Bartels* (cit. *Lenk*).

The radiological appearances may be divided into three groups:

(1) The lung infiltration and lymphatic glands are separately visible.

(2) Only the glands are visible. The appearances are then those of a mediastinal tumour.

(3) The lung infiltration is visible, but the glands cannot be directly seen.

In the first case the diagnosis of bronchial carcinoma is usually easy.

In the second case, in which only the glands are visible, the differentiation from a mediastinal tumour, lymphosarcoma, or lymphadenoma may be difficult. The age of the patient and the symptoms, and the result of radiation, may be extremely helpful. Lymphosarcoma is stated (*Lenk. Holfelder*) to be more radiosensitive than lymphadenoma. Carcinomatous glands are very insensitive, but will usually react to heavy dosage.

Some help may also be obtained by observation of the position of the glands. Lymphadenoma tends to affect the tracheal and tracheobronchial glands first, but may be present in the retrosternal and bifurcation glands later. Carcinoma tends to affect the lower groups first, but may also be found in the retrosternal and paratracheal glands in the later stages. Unilateral involvement of the glands, when the mass is large, is in favour of carcinomatous metastases, since large lymphadenomata are almost always bilateral.

Thymus tumours may be impossible to differentiate from carcinomatous glands on a single examination. They may be unilateral, but if large are generally bilateral. They tend to extend upwards into the neck, and sometimes infiltrate the sternum; they are more radiosensitive than carcinomatous glands.

Aortic aneurysm may be simulated by carcinomatous metastases involving the upper mediastinum, but as a rule difficulty only arises when the aorta is completely embedded in tumour. Careful examination, including a barium swallow, enables the different parts of the aorta to be seen through, or at the edge of, the mass, and to be differentiated from it. If the calibre of the aorta is everywhere normal, aneurysm may be excluded. An aneurysm of one part of the aorta is usually accompanied by some general dilatation of other parts. Thus in aneurysms of the arch, some dilatation of the ascending aorta is present, and vice versa ("Thoma-Kienbock rule"). Lobar collapse may result from pressure of an aneurysm upon a large bronchus, but this is rare.

In the third case, i.e. when there is no lung mass, and no projecting hilar or mediastinal tumour, the presence of the glands may sometimes be detected by careful investigation of the retrosternal and retrocardiac spaces and of the bifurcation of the trachea, and be indicated by the following signs:

- (1) Displacement of trachea.
- (2) Displacement or narrowing of the aësophiagus.
- (3) Phrenic paralysis (elevation of paradoxical movement of the diaphragm).
- (4) Evidence of bronchial stenosis, inspiratory mediastinal displacement.

In rare instances the mediastinal displacement may affect only the anterior mediastinum, producing an inspiratory "hernia" in the retrosternal weak space. Less instances a case of this in which the remainder of the mediastinum is rigid as a result of the glandular involvement. Sometimes the bronchial stenosis is valvular, resulting in relative increase of translucency of the affected lung in expiration as compared with the other lung (Fig. 262).

Many cases of bronchial carcinoma arise close to the tracheal bifurcation and immediately spread to the glands of the bifurcation and the mediastinal glands. In their earlier stages such glandular enlargements may entirely escape radiological investigation. Later they may form big masses and project to one side, or both, or give rise to disseminate lesions in one or both lungs. The shadow of the glandular enlargement appears first upon the affected side at hilar level, and is usually much more evident on that side, and may remain

apparently confined to it throughout the course of the disease. This apparent unilateral enlargement, together with the non-involvement or late involvement of the upper group (paratracheal and tracheal), helps to distinguish primary carcinoma from secondary carcinoma, and the primary mediastinal tumours of the lymphoblastoma group. It must, however, be remembered that a visible projecting gland seen in the postero-anterior view may not be the only metastasis, and that considerable metastasis may be present around the tracheal bifurcation without any X-ray signs. Evidence of tracheal compression or deformity must be sought by hard films and oblique views. Careful examination of the barium-filled œsophagus, especially in the right oblique position, may reveal early evidence of enlarged tracheobronchial glands before these are large enough to project from the mediastinal shadow in the normal postero-anterior film. *Evans* has pointed out that the indentation normally seen just below the aortic impression is due to the left bronchus, and not to the pulmonary artery, which is not immediately related to the œsophagus. He also points out how the enlarged glands in close relation to the bronchi are likely to deform the œsophagus at this level. Carcinomatous glands above the left bronchus indent the left border of the œsophagus and displace it to the right; in the right oblique view the œsophagus is displaced and angulated towards the spine. Glands above the right bronchus displace it towards the left. Enlarged bifurcation glands may displace it backwards. The special significance of the group of glands at the junction of the innominate veins on the right side very near the phrenic nerve in producing phrenic paralysis, and of the "aortic group" on the left side in producing left recurrent nerve paralysis, has been pointed out by *Lenk*.

Left recurrent nerve paralysis is, of course, common in aortic aneurysm—and may also occur in tuberculous apical fibrosis. The writer has observed this in one case. Phrenic paralysis has also been found to result from tuberculous adenopathy (*Arnstein*).

The progress of carcinoma, as considered in the above account, has been visualised as an advancing spread from one centre (*a*) in the bronchial lumen, (*b*) lungwards, (*c*) hilumwards, with the factor of atelectasis sooner or later in evidence. This atelectasis may be segmental, lobar, or total, according to the size of the bronchus which is blocked, and may occur gradually or slowly. Total atelectasis of a lobe or lung usually results in the blocking of the whole supply at one time, but it is not uncommon to see segments of a lobe successively affected, as if the spread of the bronchial tumour has involved the orifices of the neighbouring bronchi in turn. In rarer instances whole lobes may become successively collapsed; thus an opacity which has long remained confined to the upper lobe may affect the lower lobe in its turn.

In the literature the disease is divided into "hilar" types and "lobar" types, according to the site at which the principal manifestations are present at the time of examination. It is better to retain the term "lobar" for t

tumours which apparently take their origin from lung tissue or a small peripheral bronchus, and grow into nodular or segmental forms. These do constitute a special type which have certain clinical and radiological characteristics.

An examination of the writer's series of primary bronchial carcinoma, comprising over sixty cases, shows that approximately one-third showed only a circumscribed nodular enlargement, one-sixth had a hilar tumour with partial atelectasis of one lobe, one-quarter had a lobar atelectasis segmental or total, without visible hilar mass, and one-sixth had an isolated nodule in the lung of the lobar or parenchymatous type.

NODULAR FORM OF BRONCHIAL CARCINOMA

Carcinoma arising from a small bronchus in the lung parenchyma penetrates the bronchial wall and often grows as a rounded nodule, preserving a well-defined outline, until it has reached a considerable size.

Clinically these tumours are distinguished by their mode of onset. Dyspnoea is not an early symptom, and may be absent. Pain is rather ill-defined in character, but continuous and constant in position. Weakness and loss of weight, or sudden hæmoptysis, are the symptoms of onset.

The most frequent sites are the posterior segment of the upper lobe and the apex of the lower lobe. Occasionally they occur in the basal part of the lower lobe.

When arising in the apex of the lower lobe they are often paravertebral and overlap the hilum in the postero-anterior view; they may then be mistaken for hilar tumours.

Course.—The tumour usually grows rapidly, and radiograms show definite increase in size in a few weeks. They are usually very malignant; the average duration of life is about eight months; whereas the average duration of life of a patient with a bronchial carcinoma of a main bronchus is a little over two years (*Tuttle and Womack*). If near an interlobar fissure, it causes the latter to bulge, and an interlobar empyema may be simulated. The tumour rarely spreads across the interlobar fissure, but remains confined to the lobe in which it originates. It may grow so large that it entirely fills an upper lobe; before this happens, the tumour sometimes shows an infiltrating edge.

When the lobe is completely filled with tumour substance the interlobar pleura may be strongly bulged towards the adjacent lobe. This is most readily appreciable in right upper lobe tumours. Although the tumour practically never spreads by continuity across the interlobar pleura, it may invade other lobes via the hilum. The outline of the tumour may be nearly completely spherical, but more commonly show some degree of nodulation, and in a few instances satellite nodules may be found in its vicinity (*Carman*). *Kirllin and Paterson* state that in their series of fifteen proved cases the edge lacks the sharp demarcation of a nodular metastasis. The writer has, however, seen

many examples of nodular carcinomata of the lung parenchyma retaining their sharply defined contours. though as large as a grapefruit. On the other hand, smaller ones may become invasive, and rapidly extend through the lung towards the hilum, or the surface, or both. This may occur in the absence of demonstrable infection. In other cases, infection may be the cause of much

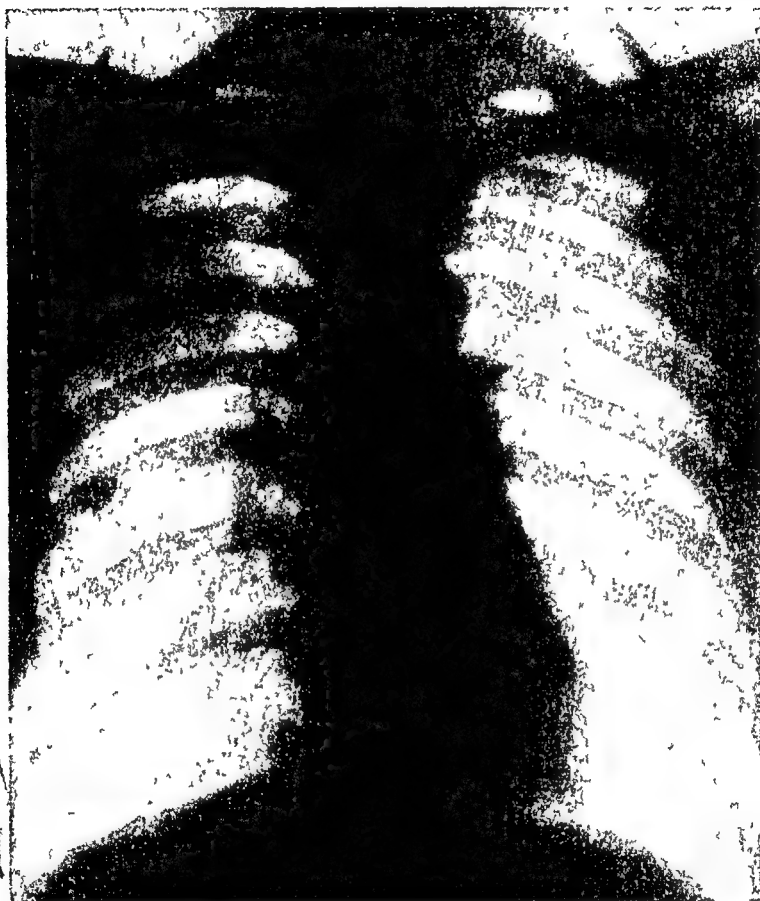


FIG. 263.—Bronchial carcinoma—nodular type. Primary arising in a small bronchus. Confirmed by autopsy 9 months later.

of the surrounding pneumonic shadow, as has been shown by *Kirklin* and *Paterson*, *Lenk* and *Ross Golden*, with post-mortem verification.

The nodular form is usually unaccompanied by radiological evidence of metastases in the hilar or mediastinal glands. Occasionally a band of infiltration is seen uniting it to the hilum. Atelectasis is rarely seen, but may occur after invasion or compression of a large bronchus by a parenchymatous tumour. Infection of the pleura may occur at any time in the course of the disease, with increase of the pain, dyspnoea, and pleural effusion. Direct spread through

pleura to a rib may occur, and evidence of local rib involvement should always be looked for ; this sign is strongly presumptive evidence of malignancy, and a contraindication to lobectomy.

Multiple nodules in primary carcinoma of a bronchus are rare. They are due to lymphatic dissemination, and occur in the same lung. Very rarely

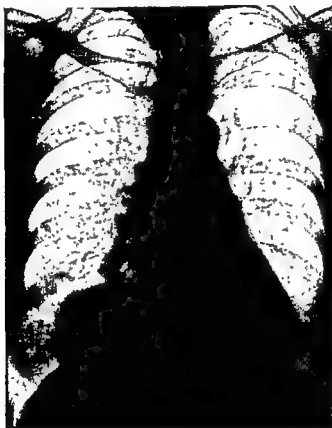


FIG. 264.—Primary carcinoma, arising in a small bronchus in right lower lobe. F. 57. Six months' history of repeated daily vomiting, latterly of cerebral type. Death from cerebral metastasis.

invasion of veins of bronchial arteries or of the heart itself may lead to hæmatogenous dissemination.

CAVITATION IN BRONCHIAL CARCINOMA

Cavitation may occur at any stage of bronchial carcinoma. It may be due to necrosis of the tumour or be bronchiectatic.

Necrotic Cavitation

When a lobe is grossly infiltrated by tumour, central necrosis and cavitation may occur without any radiographic evidence. The cavity sometimes remains filled with tumour detritus and secretions, and owing to the stenosis of the



FIG. 265.—Specimen: Squamous epithelioma of lung. The growth is necrosing, forming a large cavity into which project irregular masses of growth. The wall is composed of a thick layer of growth. Such cavities may fail to show in a radiogram: sometimes the thick irregular wall and projecting nodules can be recognised. (Pathological Museum, Victoria University, Manchester.)



FIG. 266.—Thick-walled cavernous neoplasm of lung. Erosion of the overlying rib was present. Biopsy: squamous epithelioma.

drainage bronchus, insufficient air enters the cavity to be seen in the radiogram. Lipiodol may, for the same reasons, be unable to enter the cavity. In many instances, however, careful inspection of the opaque area will show one or more translucent areas, due to cavitation. Necrosis in the nodular parenchymatous tumours is fairly common. The result is a cavernous neoplasm,



(a)



(b)

FIG. 267.—(a) Bronchial carcinoma, cavernous type M 58. Six weeks' haemoptysis, loss of weight, weakness, no dyspnoea, sputum not purulent or foul (b) Lateral view. Growth occupied axillary and posterior apical segments of right upper lobe

characterised by a sharply defined, occasionally lobulated outer contour and a thick and irregular wall, sometimes showing nodules projecting into the lumen of the cavity (Fig. 265). Fragments of broken-down growth may sometimes be found in a malignant cavity. Such sloughs are not diagnostic of malignancy.

They may be found in lung abscess at operation. The differentiation from a lung abscess is at times impossible, and many mistakes have occurred. A lung abscess is, as a rule, surrounded by a zone of pneumonitis, gradually fading off at the periphery, and rarely shows a well-defined outer edge. Multiple lesions are more often due to abscess, and are easily distinguished from multiple metastatic nodules with central necrosis. But no absolute rules can be laid down in the differentiation of these conditions; it would be easier to indicate many points of similarity between them; for a cavernous neoplasm is, in fact, a lung abscess, the wall of which is composed of neo-



FIG. 268.—Cavernous type of bronchial carcinoma. M. 58. Several months' dyspnoea, loss of weight, cyanosis, bronchitis. Autopsy: Carcinoma occluding right lower main bronchus; malignant abscess of lung, with irregular necrotic walls formed by growth. Bronchiectasis.

plastic tissue instead of inflammatory tissue. Thickness of the wall is no criterion, for this simply depends on the relative size of the necrosed centre and the, as yet unaltered, peripheral part. Thus we may find necrosing nodular cancers of the lung with walls of any thickness, diminishing with time until necrosis has caught up with growth. The wall may then be no more than

a few millimetres in thickness, and the lesion may resemble a chronic abscess or even a tuberculous cavity.

Sharp definition of the outer wall is a rather more reliable sign, but does not exclude an abscess. Old chronic abscesses may have well-defined walls, and re-

cent abscesses in the pre-necrotic stage may be sharply defined. Fig. 165 shows an abscess which three weeks later ruptured into the pleura, in the early stage it is radiologically indistinguishable from a carcinoma. Most stress must be laid upon the rate of change of the appearance, which is rapid in recent abscesses, relatively slow in neoplasm. The clinical history and signs, and character of the sputum (which is sometimes not foul in cavernous neoplasm), usually decide the question.

The difficulty of deciding whether the X-ray appearances are due to a malignant or non-malignant condition is increased by the fact that there may be a "para-malignant" abscess in the lung, due to the breaking-down of an area of infective pneumonitis adjacent to the actual growth (see Fig. 269).

Chaoul and *Greiner* believe that tomography will sometimes distinguish between abscess and growth, the surrounding lung being homogeneous in growth (atelectasis) and fading off towards the periphery, or extending outwards in strand-like processes, while in abscess the surrounding lung has a mottled appearance.

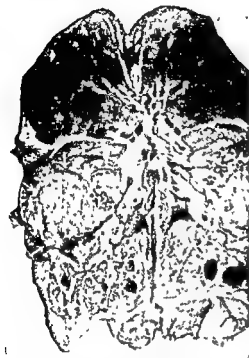
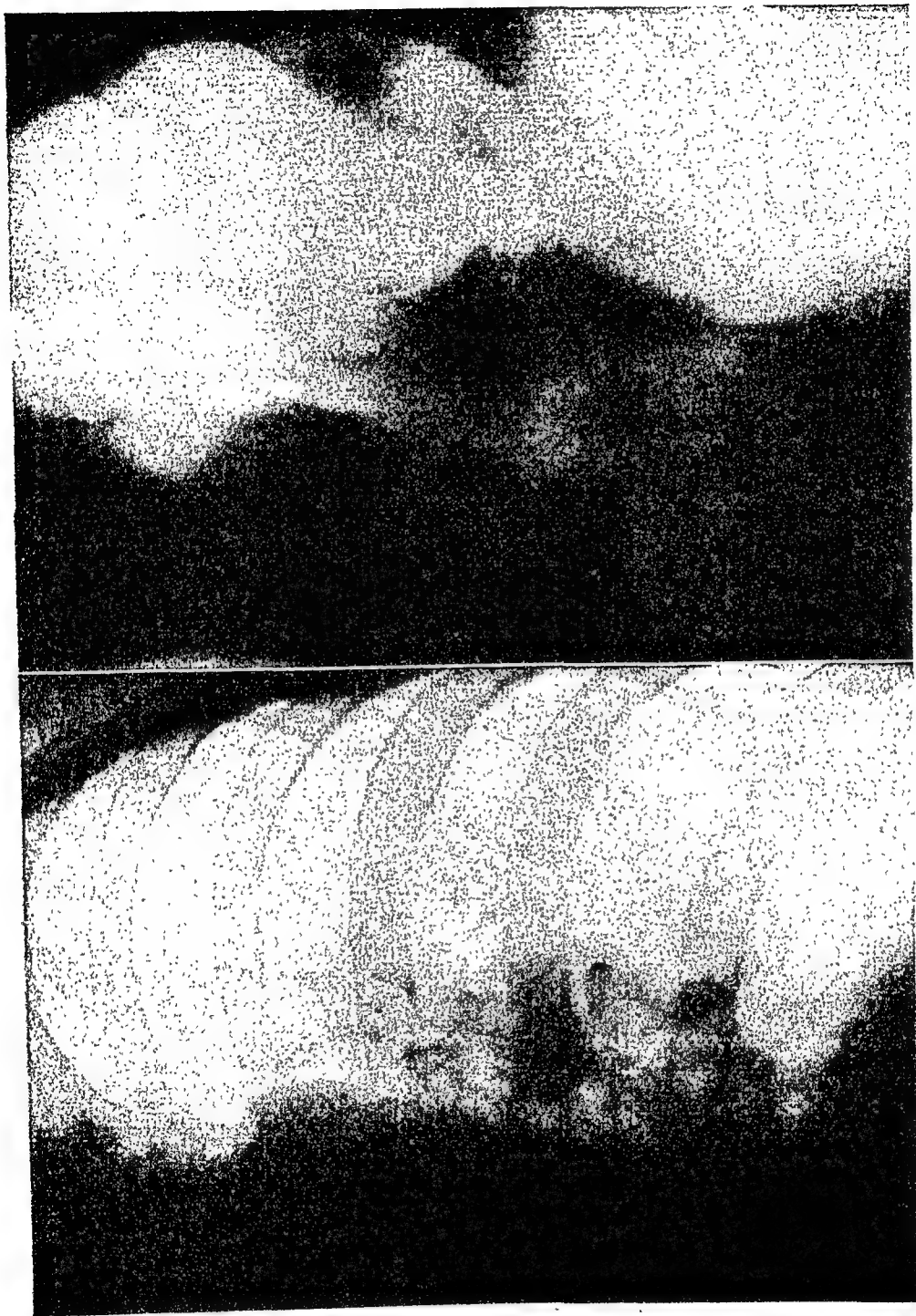


FIG. 269—specimen Bronchial carcinoma, with cavitation. There is a large "para-malignant" cavity (arrowed), the walls of which are not entirely composed of growth but to a great extent of infected lung. Many bronchiectatic cavities are present in the lower lobe (Pathological Museum, Victoria University, Manchester.)

processes, while in abscess the surrounding lung has a mottled appearance.

Bronchiectatic Cavitation

Bronchiectatic cavitation follows bronchostenosis, and is only seen in carcinoma of the larger bronchi. The usual sequence of events is bronchial stenosis or occlusion, atelectasis, infection, perforation of the bronchial wall with multiplied ragged cavities. These cavities are nearly always hidden by the atelectasis, though careful search will occasionally demonstrate translucent areas in a collapsed lobe. In one personally observed case a homogeneously



Tomogram of the same case; the abscess shows clearly. Its walls are irregular. A nodule projects from the hilum into its medial side.

FIG. 270.—Bronchial carcinoma with malignant abscess. Straight radiogram. The abscess cavity can be seen only with difficulty.

atelectatic left upper lobe was six weeks later found to be riddled with cavities of an inch or more in diameter.

MALIGNANT PLEURISY IN BRONCHIAL CARCINOMA

The pleura may be directly involved early or late in the course of the disease. Multiple small pleural foci, or a few larger nodular foci, may be present, but are themselves invisible in the radiogram. The commonest result is pleural effusion, often hæmorrhagic, which tends to obscure the actual growth, particularly when this is basally situated. The diagnosis of neoplasm is, however, always suggested if the heart and mediastinum are not displaced towards the opposite side (as a consequence of underlying atelectasis). An attempt to uncover the tumour by withdrawing the effusion by simply tapping often fails, because even the withdrawal of large amounts of fluid fails to alter the level of the effusion appreciably. The chest wall falls in; the space to be occupied by the fluid is thereby diminished, and the residual fluid is still found spread over the base of the lung.

Better success attends a gas replacement of the fluid. The writer, with *R. Williamson*, investigated a number of cases of effusion air replacement in 1923. This method enables a far more complete drainage to be effected, and a neoplasm previously hidden by fluid may be thus brought to light. If, however, the lobe is much collapsed, it may be too opaque to prevent any differentiation between lung tissue and the growth.

It is sometimes possible to demonstrate a hilar growth after partial gas replacement of the effusion by lying the patient on the affected side, and taking a postero-anterior radiogram in this position. The fluid gravitates to the parietal wall and uncovers the hilum region, where the growth may then be found. Less commonly a peripheral growth may be thus demonstrated with the patient lying on the sound side. As a rule, even these manoeuvres fail to clear up the diagnosis.

A few of these cases showed evidence of extensive pleural adhesions; the fluid then tended to collect in pockets of adhesions, showing multiple fluid-levels throughout the hemithorax. This occurred principally in extensive growths in which the entire lung was opaque. Such fluid-levels are occasionally seen after ordinary paracentesis, when air has accidentally entered the thorax. In the light of experience with air replacement, the writer feels certain that they are evidence of pleural adhesions; after tapping or air replacement malignant effusions recur rapidly: the original quantity of fluid may again be present in a few days.

The effusions are not always malignant. They may be simple transudates, or purulent. In the former case they may become absorbed. Pathological examination of the fluid is often of great value. It is often hæmorrhagic and may contain malignant cells. These are more readily found if the fluid

is centrifuged after the addition of blood-serum. The fibrinous clot collects the cells, and these are seen in histological sections of the clot (*Christie*).

The writer has observed a case which exemplified the variable radiological appearances which may be given by a neoplasm under different conditions.

The first examination showed a hilar tumour; this was followed by an effusion: the hemithorax was then completely opaque, and the mediastinum bulged towards the opposite side. After tapping, the effusion became absorbed, and the hilar tumour was again seen, larger than before. After a further interval atelectasis supervened, and finally a further effusion. The hemithorax was again completely opaque, but on this occasion, owing to the atelectasis, the displacement of the mediastinum was to the side of the lesion.

In the first opaque stage distinction from a simple effusion would have been impossible; in the second distinction from fibroid lung would have been difficult, but for the previous knowledge of the existence of hilar growth.

Lenk and Kerley have pointed out that if an effusion is masking an atelectatic lobe, and if phrenic paralysis is also present, the diaphragm on the affected side is depressed by the effusion. On the right side this will depress the liver, which is felt below its normal level; on the left side it will depress the gastric air bubble. Under these circumstances it is possible to diagnose the presence of effusion in doubtful cases.

SUPERIOR PULMONARY SULCUS TUMOUR

A pulmonary carcinoma may arise at the extreme apex and show a rounded supraclavicular shadow, extending later below the clavicle. Such a tumour grows rapidly and erodes the first rib and spine, and produces characteristic pressure signs upon the brachial plexus (VIII Cervical, and I and II Thoracic), causing pain in the arm and wasting of the hand. If the cervical sympathetic is also involved, Horner's syndrome (ptosis, miosis, enophthalmos, and anidrosis) is also present (see Fig. 271).

This tumour has been described by *Pancoast*, who saw seven cases, as the "Superior pulmonary sulcus tumour." He believed that they arose from branchial clefts. *Connolly* in this country has published a similar case with autopsy findings. The histological structure was that of an adenocuboidal tumour, almost certainly a primary carcinoma of the lung apex.

Not all such apical tumours are, however, of pulmonary origin. The writer saw a case of a large rounded subclavicular tumour in the left apex projecting down into the lung field with a rounded lower border, which was an enlarged cervical gland, in a case presenting multiple glandular metastases, the primary growth being a teratoma of the testis. The upper pole of the mass was palpable in the neck. There was no rib destruction. The trachea was markedly displaced, but the mass did not reach down to the main bronchus, nor did it involve the tracheobronchial glands. Two other cases of secondary malignancy

nancy of glands in the neck showed a large apical tumour, extending downwards into the apical region of the thorax, with erosion of the first rib and brachial plexus nerve involvement. Involvement of the brachial plexus and of the cervical sympathetic, the latter producing Horner's syndrome, is not



FIG 271—Carcinoma occupying the right apex "Superior pulmonary sulcus tumour" of Pancoast. Note erosion of first to third upper ribs. F 76. Complaint: pain in shoulder and arm. Adenocarcinoma of peripheral origin.

confined to the so-called "sulcus tumour," but may result from tumours of the nerve sheaths or of the spine, tumours and malignant lymph nodes in the neck, trauma, aneurysms, mediastinal tumours, and pulmonary tuberculosis.

Since *Pancoast* described the "Superior sulcus tumour" in 1932, many writers have contributed examples of tumours in the same situation (*Fried, Jacox, Steiner and Francis, Clarke, Graef and Steinberg*).

Jacox's case was an adenocarcinoma, with mucin-forming cells, *Clarke's* an

epidermoid carcinoma adherent to, but not in, the lung apex.

Graef's case was an invasive carcinoma, extrapulmonary; histologically it showed in some places an alveolar structure, in others a squamous epithelial arrangement.

Apical fibroma of the pleura, neurofibroma, hæmatoma, tuberculous abscess of the rib, sarcoma, and encysted apical empyema are other conditions which may have to be considered in the differential diagnosis, since they may produce similar shadows.

THERAPEUTIC TESTS IN BRONCHIAL CARCINOMA

The result of deep X-ray therapy in a case showing complete atelectasis of one lung is occasionally astonishing. The administration of an adequate dose

to the affected bronchus and the tracheobronchial glands is in some cases followed by a complete disappearance of the opacity.

The emphasis which in the literature is laid upon the factors of infection and pleural thickening in producing a radiological picture of total unilateral obscurity often engenders a feeling of pessimism; the radiological appearance is apt to suggest that the underlying lung is "dead," possibly riddled with infection and enclosed in a layer of grossly thickened pleura. Such a condition may, of course, exist, and the writer has seen it at autopsy. But it cannot be inferred from the radiogram alone that this has occurred: X-ray treatment, by reducing the size of the endobronchial tumour may quite rapidly restore the patency of the bronchus, for a time at least. The lung re-aerates, and allows the hilar mass to become visible, sometimes much reduced in size.

This therapeutic effect is not obtained without heavy radiation, and if it is decided to employ it, half-measures are useless. In several cases, however, the writer has observed complete resolution of a total atelectasis, which had resulted from obstruction of a main bronchus. In the case shown in Fig. 261 radiotherapy had been employed. The atelectasis later recurred and the patient died of his malignancy.

PRIMARY SARCOMA OF LUNG

It has been doubted by many authors whether primary sarcoma of the lung occurs. The histological distinctions are not always clear. One autopsy series (*Lenz cit. Lenk*) show only one primary lung sarcoma but 57 primary carcinomata in 5,600 autopsies. *Lenk* saw 100 verified carcinomata, but only one sarcoma. The writer has seen only one verified primary sarcoma (fibrosarcoma) (Fig. 272).

The tumour arises in the peribronchial or pulmonary connective tissue, or in the lymph glands. It may be round or spindle celled, or a lymphosarcoma. It usually forms a rounded nodular well-defined tumour or may be encapsulated. It may involve a whole lobe and, like carcinoma, tends to respect the interlobar pleura. Bronchostenosis is rare. Cavitation seldom occurs. Metastases are uncommon, but may be found in the liver. Pleural involvement and hæmorrhagic effusion are relatively common.

HYDATID CYST OF THE LUNG

Though of parasitic origin (*Tænia echinococcus*), hydatid cysts may for radiological purposes be considered among the tumours of the lung. It is not a rare disease in this country. *Brailsford* comments upon the ease with which slaughterhouse conditions might allow transmission from sheep, frequently infected, through dogs to man. It is very common in Australia and New Zealand. In Iceland it has been carefully studied by *Claessen*. In man the path of infection is through the alimentary tract: the embryos set free from

the ova usually reach the liver through the portal vein and develop there, but may pass into the general circulation and thus reach the lungs.

Déré, by experimental injection into the bronchi, has shown that it may develop from inhalation; he has also shown that a primary cyst, if fertile, may reinfect the host after rupture, without an intermediate host. *Hebert* has, however, shown such secondary cysts to be rare.

Of all hydatid cysts, 75 per cent. occur in the liver, and only 8-12 per cent. in the lung. Many cases of "pulmonary" hydatid cysts, diagnosed on the



FIG. 272.—Sarcoma of lung (operation)
F. 24 Eight weeks' pain in chest and loss of weight Slight cough

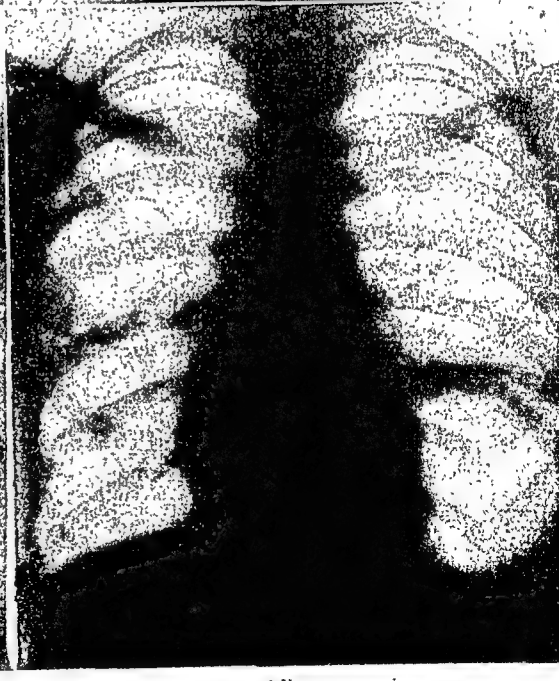
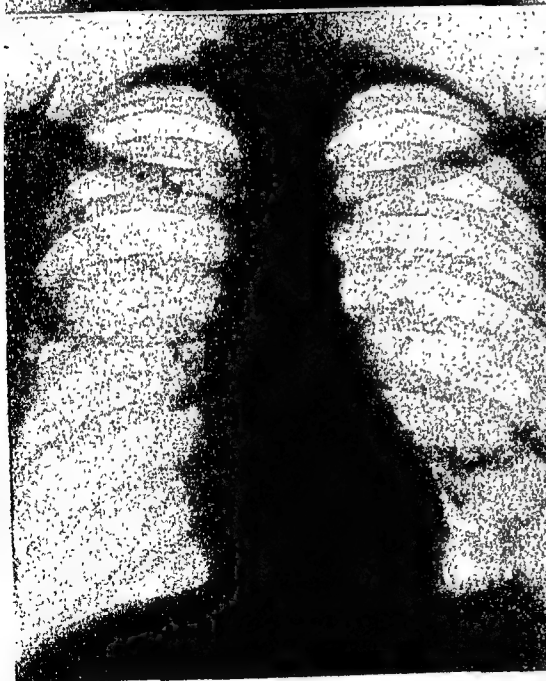
Lateral view Growth confined to left upper lobe, bulging the main fissure

evidence of expectoration of "grape-skin" sputum, are really instances of liver cysts which have ruptured into the lung. Bile may be present in the sputum in such cases, and give a clue to the origin of the cyst.

Radiology has greatly helped in the diagnosis of pulmonary hydatid disease. It must be supplemented by immunological tests, precipitin, complement fixation (*Bordet-Gengou*), and intradermal (*Casoni*). The latter consists of an intradermal injection of prepared sterile hydatid fluid. The complement fixation test is said to be positive if the cyst has ruptured, negative if it is unruptured or dead. The *Casoni* test is positive in over 90 per cent. of uncomplicated cysts. Eosinophilia is often present. Paracentesis should be avoided. Clinically the disease may show a prolonged latent period, with

(a)

(b)



(c)

(d)

FIG. 273.—(a) and (b). Hydatid cysts in right upper and left lower lobes. (c) Same case after removal of cysts : the spaces previously occupied by the cysts are shown as air containing cavities surrounded by indurated lung tissue. (d) Four months later : the cavities have almost disappeared.

hæmoptysis, stitch-like pain, and cough, and may simulate tuberculosis, pleurisy, or neoplasm. The cysts do not grow very rapidly in the lung, but can reach a diameter of 2 inches within a year (*Tudor Edwards*).

Radiological Appearances.—The right lung is more frequently affected than the left (*Claessen*). The classical appearance is that of a circular shadow, sharply defined, with no reaction in the surrounding lung parenchyma. It should, however, be noted that this classical circular form is not always present. Often it is oval or ovoid. It may become more oval on deep inspiration ("Escudero-Nemenow" sign). It may be modified (a) by the presence of the cyst in a confined region of the thorax; (b) by mutual pressure of two cysts; and (c) by position adjacent to parietes, interlobar pleura, or mediastinum. In this case the cyst may show one side flattened and continuous with the parietal, interlobar, or mediastinal shadow. If parietal, it will then have to be distinguished from pleural fibroma, sarcoma, or parietal encysted effusion. Occurring near the mediastinum, it will simulate aneurysm, mediastinal tumour, or dermoid. In the lung it may resemble an interlobar effusion. *Fleischner* has met with a case which was actually interlobar. After rupture into a bronchus and partial evacuation, it will simulate an abscess. Irregularities of contour, due to portions of the endocyst projecting above the fluid, may be seen. *Anderson* has demonstrated the separation of the cyst membrane from the surrounding pericyst after puncture. Some air had entered the cavity. His radiogram showed a horizontal fluid-level; above this a circular air shadow demarcated from lung by the arched shadow of the pericyst. Below the latter was a second arched line, due to the collapsing cyst membrane. After death of the parasite the cyst loses its sharp definition in the radiogram, and may calcify. The calcium is deposited superficially in the fibrous ectocyst. If the cyst suppurated, the well-defined contour is also lost.

After complete evacuation, or after operative removal, a circular air-containing space may remain for a considerable time, surrounded by a thin wall of condensed lung tissue, resembling a chronic tuberculous cavity, or congenital cyst. It gradually disappears.

In a case published by *Bronley*, the cyst, which was in contact with the anterior chest wall, showed a very irregular shape in the antero-posterior view—resembling the wing of a butterfly, possibly due to some effect of local pressure. In a case observed by the writer, an irregular triangular shape was due to collapse of the cyst after rupture into a bronchus. A hydatid of the liver, if superficial, produces a dome-shaped local elevation of the diaphragm into the thoracic cavity. If the cyst is deeply placed in the liver, the diaphragm is raised as a whole. Artificial pneumoperitonæum may assist in the diagnosis; in a case observed by the writer, the shadow which filled the lower two-thirds of the right hemithorax was in this way proved to be due to an intra-hepatic cyst.

Anderson draws attention to a radiological sign which may be present in



FIG. 274.—Hydatid cyst, lateral view.
Operation, cure.

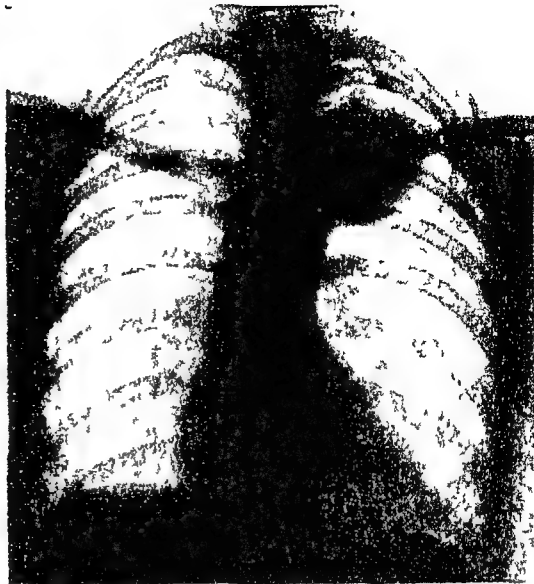


FIG. 275 —Chondroma, arising from an inter-
vertebral disc, and projecting into left lung.
Biopsy.

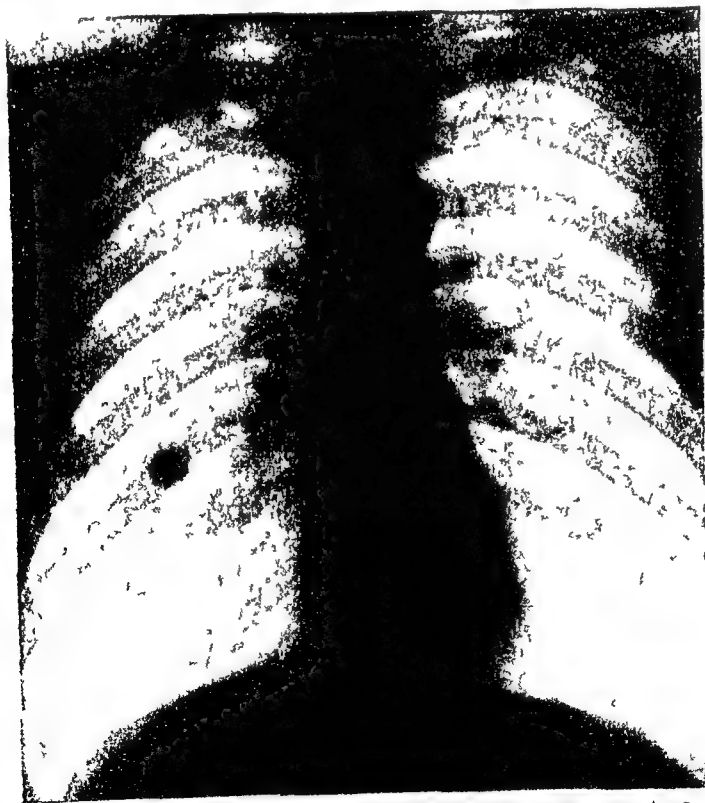


FIG. 276.—Chondroma of the lung. The centre of the tumour mass is densely calcified.

cases of hydatid cyst of the liver, namely, increase in the lung markings extending upwards from the liver to the lung root. He suggests that it may be due to an irritative lymphangitis.

Hydatid cysts of the lung may occur in any situation. It has been suggested that they are more often posterior in site, but cases have been published showing apical, dorsal, anterior, basal, or mediastinal positions, and they often occur near the periphery.

Hebert has collected a few cases of secondary bronchogenic cysts, resulting from reinfection of the host from a primary ruptured cyst, which may be in liver or bone. These show multiple rounded nodules resembling metastases in the lung. Multiplicity is common. As many as seven or eight cysts have been observed grouped like a bunch of grapes around the larger bronchi (*Tillier*).

SIMPLE TUMOURS OF THE LUNG

These are very rare. Chondromata, lipomata, and fibromata have been reported.

Chondromata of the lung have been described by *Hirley* and *Simpson*. The radiogram shows a well-defined, rounded, or slightly lobulated shadow in the lung, which may show a dense calcification in the interior. *Kerley* has recently seen two chondromata, one arising from the mediastinum on the left side, and one in the right lung. Of these, the first was verified by operation.

A case of fibroma of the lung is quoted by *Sante*. It probably arose as a fibrous deposit around a suppurative focus, since a lung abscess cavity was found in the centre surrounded by many layers of thick fibrous tissue.

CHAPTER XXXII

METASTATIC TUMOURS

PATHOLOGY

Langenbeck, in 1840, produced growths in the lungs of dogs, by intravenous injection of fresh tumour material, and *Weil*, *Iwasaki*, and *Takahashi* have verified these experiments in rats and mice. Many of the tumour cells injected into the blood-stream perish in the lungs, and their degenerated remains have been demonstrated histologically in the pulmonary vessels. The more highly differentiated squamous-celled and adenocarcinomata fail to grow in the lungs, while less differentiated and rapidly growing tumours grow more readily and give more "takes." Secondary neoplasms of the lung may arise in the following ways (*Willis*) :

A. Direct Non-metastatic Invasion

(i) By contiguity. (ii) Via lymphatics. (iii) Via pulmonary arteries.

B. True Embolic Metastases

(i) Via pulmonary or bronchial arteries. (ii) Via bronchi (aerial metastasis).

A. Direct Invasion

(i) INVASION BY CONTIGUITY is seen in primary intrathoracic or cervical growths, e.g. thymus, œsophagus, or thyroid. It may occur from mediastinal lymph-nodal growths. Breast carcinoma and pleural carcinoma may spread directly to the lungs. Abdominal tumours may invade the lungs directly through the diaphragm, or reach them from a cancerous thoracic duct.

(ii) INVASION VIA LYMPHATICS.—Deposits of cancer in hilar and tracheo-bronchial glands obstruct the normal efflux of lymph from the lungs and then extend by retrograde permeation of the peribronchial and subpleural lymphatics; this extension may be limited to the hilar region, or extend widely through the whole lung in fine tumour cords, accompanying bronchi and veins, forming a delicate network in the lung parenchyma; and ramifying in the subpleural lymphatic plexus. On this network of tumour cords through lung and pleura, nodular thickenings may occur at the main junction points, which may grow and invade the lung. A continuous extension by permeation may be supplemented or replaced by an embolic transference through lymphatics in which as a result of local block, the usual direction of lymph flow has been reversed.

The primary tumour most often responsible for lymph-vessel cancer of the lungs is gastric carcinoma, which reaches them via the cœliac and posterior mediastinal groups of glands. Other primary tumours which may give rise to this form are carcinoma of the breast, of the lung itself, gall-bladder, and prostate. Lymphatic permeation of the lungs from a carcinoma of the tongue, though rare, has been described by *Ewing*.

(iii) **INVASION OF PULMONARY ARTERIES.**—This is rare. Intravascular tumours which have spread along the vena cava into the right heart may continue to spread intravascularly into the pulmonary arteries. This has been recorded in teratoma of the testes and intravascular chondroma. Tumour of the left auricle may grow into the lungs along the lumen of the pulmonary veins.

B. True (Hæmatogenous) Metastatic Tumours of the Lung

These result most frequently from tumour invasions of the systemic veins, sometimes from invasion of large lymphatic trunks or thoracic duct, and occasionally by invasion of the heart walls. The emboli are in all cases disseminated by the pulmonary arteries and grow in the lung. It is possible that the bronchial arteries may in some cases be the path of dissemination, and *Willis* suggests this explanation for the scattered discrete metastases in both lungs occasionally found accompanying a primary growth of one lung. The emboli in this case enter the bronchial arteries via the pulmonary veins and left heart.

Frequency of Metastatic Tumours in the Lungs

The lungs are the most frequent seat of metastatic growths for almost all malignant neoplasms, except those arising in the portal area. *Willis* believes that blood-borne lung metastases occur in approximately the following percentages in various groups of tumours. oral and pharyngeal carcinomata 30, œsophagus 20, stomach 20, intestines 15, liver 20, pancreas 20, breast 45, uterus 15, ovary 10, prostate 35, thyroid 65, kidney 75, melanoma 60, bone sarcoma 60, chorion carcinoma 70.

If all malignant growths are considered together, pulmonary metastases occur in about 30 per cent.

Gross Appearance of Metastatic Growths in Lung

These may be single or multiple. The largest number is usually seen in melanoma, sarcoma, and chorion epithelioma. Sometimes they are all of equal size; in other cases they are of different sizes, suggesting embolic inoculations of different ages. Solitary metastases are more often basal than apical.

Miliary carcinosis, in which the lungs are uniformly studded by a profusion of minute tumour nodules, is a rare form, sometimes mistaken for miliary tuberculosis. This form arises from invasion of a systemic vein of the thoracic duct.

Most metastases are spherical and sharply defined. are firm and uniform in texture. Those derived from osteosarcoma may contain osteoid or even fully calcified bone. Central necrosis and liquefaction is fairly common. though cavitation from discharge through a bronchus is less common in secondary than in primary lung tumours.

Method of Extension in Lung Tissue

Extension may occur in the lung by one of two routes : (a) intra-alveolar, and (b) interstitial.

(a) In *intra-alveolar growths* the tumour uses the alveolar spongework as a preformed stroma, extending in the lumen from one alveolus to another ; intact septa persist for some time in the peripheral part of the tumour (" pneumonia carcinomatosa "). Eventually the alveolar septa disappear. Occasionally neoplastic consolidation without loss of lung architecture occurs in patches over a widely diffused area. *simulating bronchopneumonia to the naked eye.*

(b) In *interstitial growths* the tumours spread in the *alveolar walls*. flattening the alveoli. Lymphatic permeation assists in the process of spread.

CLINICAL COURSE

If the lesions are blood-borne, they may appear abruptly and rapidly develop. If lymphatic, there is often a latent period in which the disease progresses slowly in the mediastinal and hilar glands, and repeated observation is necessary to establish the presence of extension to the chest. This is especially true in carcinoma of the breast. The often chronic course of this disease so readily observed in glands which can be seen and palpated. e.g. in axilla and supraclavicular regions, is matched by a course no less chronic in the mediastinal glands, so that the radiologist. when asked to determine whether there is evidence of intrathoracic extension (which would contraindicate radical operation). knows that his examination may fail to show glands which are actually present. In some cases there is a slight suspicion of enlargement of one hilum, and only time and repeated observations will enable the diagnosis to be made.

The presence of metastases in the chest is often associated with no symptoms at all, and they are frequently brought to light by routine radiological examination. Constant watch has to be kept on patients under treatment for carcinoma of the breast ; it is the writer's custom to examine them radiologically every month. It is usually impossible to detect metastases by clinical methods.

The most suggestive symptoms are : (1) Dyspnoea. frequently slight and only elicited by questioning the patient. (2) Loss of weight, and pallor. (3) Pain, more particularly if ribs are involved. (4) Cough may be present, but is often absent. (5) Small hæmorrhages may occur, but profuse hæmoptysis is rare, except in chorion epithelioma.

RADIOLOGICAL FEATURES

PLEURA.—Involvement of the pleura occurs in two forms. The commonest is a pleural effusion, especially in breast cancer. The onset of this is always insidious, and we sometimes notice a slight breathlessness of the patient when



FIG. 277.—Metastatic tumour of pleura, and hilar gland. M. 28
Primary was an endothelioma of the groin

speaking, before she is aware of it herself. On enquiry, she will admit that she has noticed breathlessness. Percussion and screen examination often show the beginning of an effusion which progresses rather rapidly, and always recurs quickly after tapping.

Much less frequently a pleural nodular metastasis is seen. In appearance it is similar to a localised parietal effusion, but the history is decisive. Localised pain is often present, but may be absent.

Ribs.—This type of metastasis must be noted here because of the difficulty of diagnosis. If the ribs are occupied by multiple small hæmatogenous metastases, none of which has caused erosion or expansion of the rib contours, it is hard to distinguish them

from the overlying structures. Stereoscopy, or careful filming with the patient rotated first in one direction, then in another, in order to vary the relation of lung tissue to the suspected ribs, will sometimes serve to prove that the lesions are endosteal. In other cases expansion, localised erosion, or pathological fracture, is obvious. The posterior ends of the ribs are very frequently involved.



FIG. 278.—(a) Lymphatic spread of secondary carcinoma in lung (lymphangitis carcinomatosa). Primary carcinoma of breast. Increased hilar shadows, especially right upper.



Same case one month later : extensive lymphatic spread.

MEDIASTINAL GLANDS.—Occasionally a paratracheal gland is involved. Usually, however, the tracheobronchial group is first to show visible enlargement. The shadow in the first instance is seen to the right of the trachea above the bifurcation. In the latter case, nothing abnormal is seen in the postero-anterior view until the glands have enlarged sufficiently to project into the hilar region, but examination of the barium-filled œsophagus may detect the presence of enlarged tracheobronchial glands at an earlier stage.

LUNGS.—Extension from the hilar gland may take place in many ways :

(1) As a local and rather diffuse streaming out of thickened strands into one lung

(2) As a bilateral generalised lymphangitis carcinomatosa. This is not a common condition. The fine strands gradually extend on both sides, and the lung fields become opaque. There is often marked dyspnoea at this stage, but it may be absent. Night sweats may occur. The lymphatics are found to be much thickened and permeated by solid strands of carcinoma. Small nodules may develop along these strands; in the film there will be fine condensations strung along the network of pulmonary markings. A case with post-mortem findings is published by *Schwarzmann*. The primary was in the prostate

The writer has observed this form several times in carcinoma of the breast; in bronchial and gastric carcinoma, once in prostatic carcinoma, and once in a case of adenocarcinoma of the cervical glands in which the primary was not discovered.

(3) As a localised extension of coarse nodules from the hilum. This may be unilateral or bilateral. The basal regions often show more and larger nodules than the apices. The nodules appear to radiate outwards from the lower hilum region, growing smaller towards the periphery. It is not always possible to distinguish such types from the blood-borne metastases.

(4) Large single nodule. Metastasis often takes this form. The nodule is usually basal, often rounded and quite sharply defined. It may grow to the size of a cricket ball or larger before breaking down. *Graham Hodgson* has shown an interesting case of this type which simulated a hydatid cyst. The differential diagnosis is easy if a primary can be found, but if not various appropriate clinical tests must be applied. The Wassermann reaction should not be omitted. A second observation may be necessary. Growths grow, and the increase in size of any metastatic deposit is usually evident within a very few weeks.

Multiple Nodular Metastases in the Lungs (Blood-borne).—The appearance of metastases from sarcoma of bone or other tissues, from hypernephroma, carcinoma in any part of the body, seminoma of the testis and chorion epithelioma, usually takes the form of rounded nodules. They may be single or multiple, large or small, and of unequal size. They are most numerous as a rule at the bases, but may occur anywhere. They are sharply defined at first, and usually

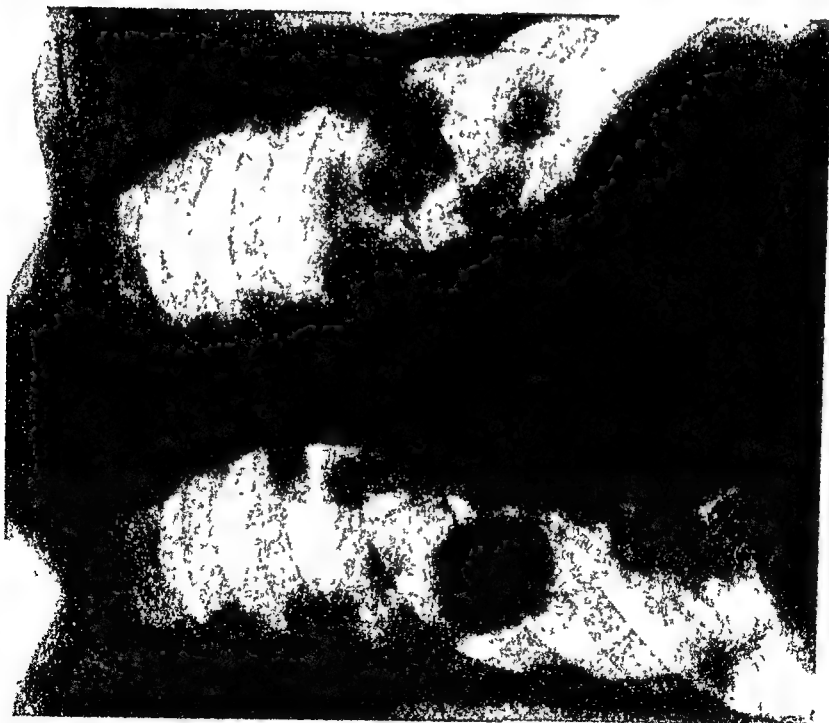


FIG. 279.—Large nodular type of metastasis in lung. The primary growth was apparently in the left lower lobe. M. 47. Cough and wasting, and pains in the limbs for three months. This type is common in sarcoma.

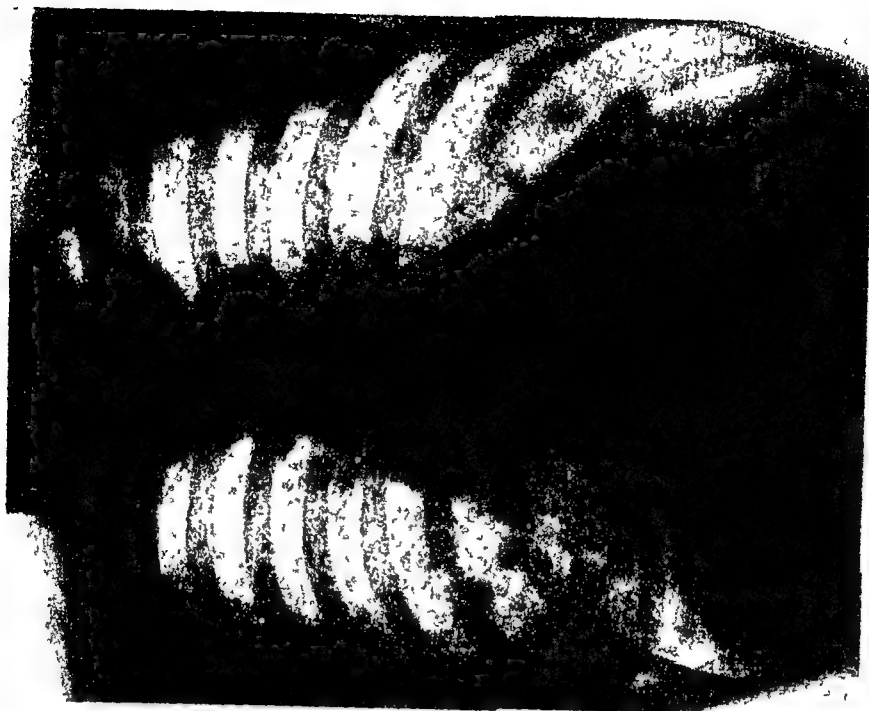


FIG. 280.—Metastatic carcinoma. Small nodular type. Primary carcinoma left breast. Note the small retracted breast shadow.

retain their defined edge even when very large and numerous. Occasionally they may become diffuse.

In a case of sarcoma of the tibia, with a single metastasis in the lung, the latter lost its rounded contour after an interval of a few months and spread diffusely through the lung. Others had in the meantime appeared and had also become diffuse. The resemblance to a tuberculous lesion was at this stage quite plausible. Blood-borne metastases show no even gradation in size from hilum to periphery. Large and small nodules may occur side by side. Though usually spherical, metastatic nodules are often somewhat oval, with their longer axis parallel to the general direction of the bronchi of the region of the lung in which they occur. They sometimes necrose centrally and form cavities. Occasionally they are not circumscribed, but are invasive from the first. Diffuse blotches of shadow then occur, resembling a rather rare type of tuberculous involvement of lung.

Miliary Carcinomatosis of the Lungs.—This is a rare form in which the lung fields are filled with small nodules or blotches of shadow due to blood-borne metastases. The appearance is not unlike that of miliary tuberculosis, but the lesions are as a rule coarser and tend to increase in size and number from above downwards, the apices being relatively free.

Frequency of Various Types of Pulmonary Metastases.—In a series of sixty cases examined by the writer, the different types of metastases in lungs and pleura were found grouped approximately as follows: One-quarter of the cases showed the coarse nodular type of lung involvement, a little more common in the lower lobes than the upper; one-quarter showed diffuse lymphatic spread from the hilum; and one-quarter pleural lesions, viz. effusion or pleural node (3 per cent). The fine nodular and fine disseminate type of secondaries were rare, together constituting about 10 per cent. In 10 per cent. the secondaries were only observed in hilar and mediastinal glands, and had not yet spread to the lung. In a few instances a lobar type occurs, producing total opacity of one lobe, usually the upper, as a result of bronchial stenosis. This condition has been observed by the writer in carcinoma of the breast and sarcoma of the tonsil; it exactly resembles primary bronchial carcinoma.

Sarcoma, hypernephroma, and seminoma are common sources of the large round "golf-ball" type of pulmonary metastasis.

The rounded type has also been noted in epithelioma of the skin and mouth and pharynx, carcinoma of stomach, thyroid, ovary, and uterus, in lymphosarcoma, and in chorion epithelioma (*Schinz*). A primary bronchial carcinoma may give rise to a multiple rounded metastasis in one or both lung fields, and the resulting picture—hilar enlargement with rounded pulmonary nodules—exactly resembles that produced by metastasis from a distant organ.

The rate of growth of metastases in the lung varies greatly. As a rule, re-examination in a few weeks shows a rapid increase in size. In some cases the growth is much slower. Subpleural nodules often give rise to a rapidly developing effusion, which soon masks the lung lesion.

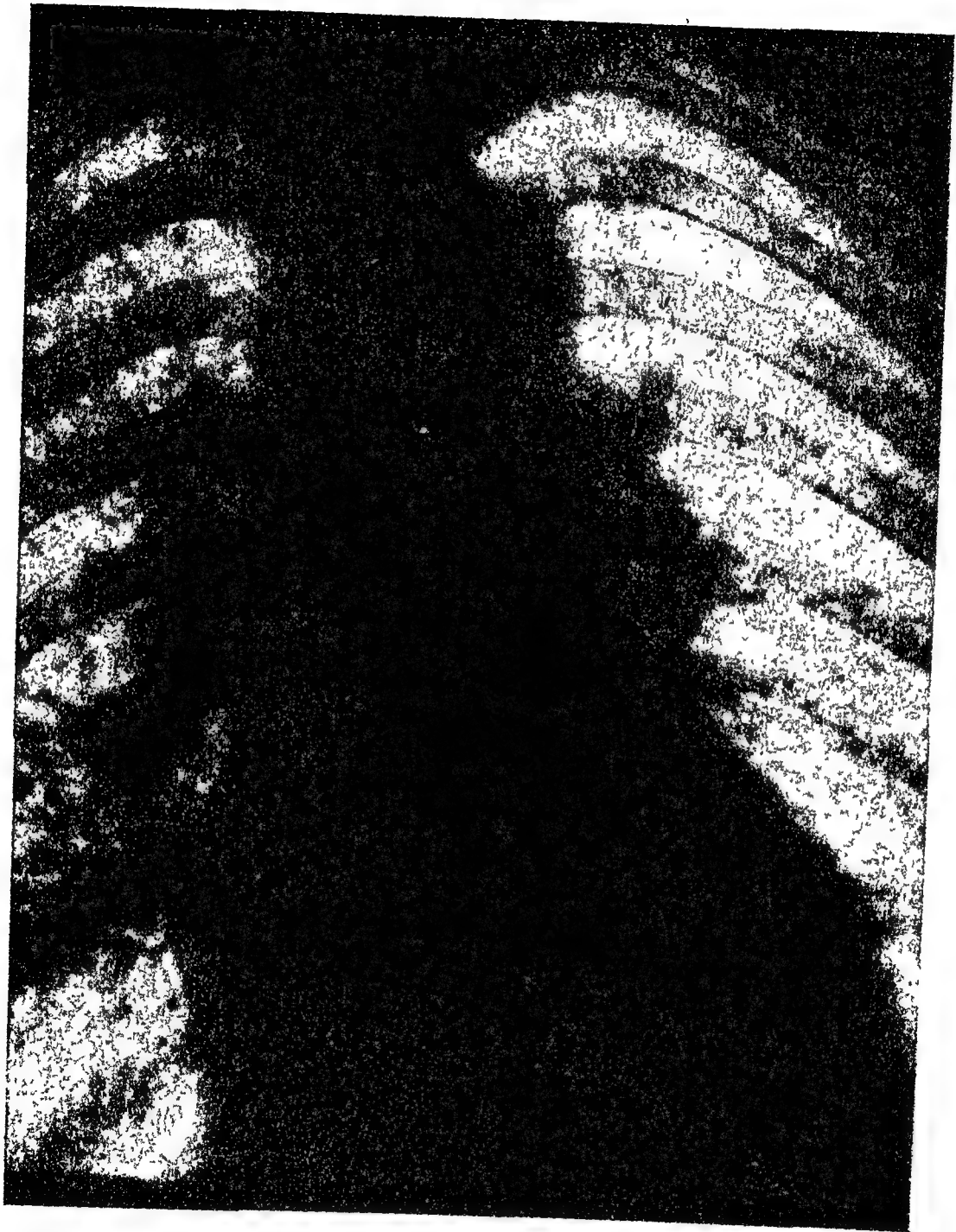


FIG. 281.—Metastatic carcinomatosis (carcinoma of prostate). "Miliary" type of lymphatic dissemination.

It is not practicable to correlate definite types of pulmonary metastases with definite primary sources. The types described above may be derived from sarcoma, or carcinoma in any part of the body. Nor is it always possible to say from the X-ray whether the spread has occurred via the lymphatics or blood-stream. Lymphatic spread is always strongly suggested when there is a regional complex consisting of hilar glands, with strands of shadow leading out into the lobe which drains into those glands, and metastases of the nodular or blotchy diffuse type in the same lobe; and serial radiograms will often demonstrate the occurrence and progression of this type of lesion.

The diffuse regional types sometimes simulate tuberculosis, but the tendency to perihilar distribution, and diminution in the size of the shadows from the hilum outwards, is unlike tuberculous involvement; re-examination at intervals of a few weeks is essential in doubtful cases. It should be remembered that tuberculosis of the lungs sometimes gives rise to rounded, rather well-defined multiple shadows.

CHAPTER XXXIII

INFLAMMATORY DISEASE OF THE PLEURA

DRY PLEURISY

IN DRY pleurisy the pleural membrane is covered with a thin sheet of exudate which becomes fibrinous, and may later become organised into fibrous tissue. Such plastic pleurisy may occur independently, or be secondary to acute disease, such as pneumonia or abscess, or to chronic disease, e.g. tuberculosis. They usually result in the formation of pleural adhesions. They are common in children ("lamellar pleurisy"). They often fail to show any radiological evidence: a careful tangential view will, however, occasionally detect a slight thickening of the pleura, in the form of an opaque lamella separating the lung edge from the chest wall. If the underlying lung periphery is affected by the inflammatory change, it loses its translucency, and the opacity is then more pronounced. The diaphragm movements may show a definite restriction. In malignant disease, particularly bronchial carcinoma, gross thickening of the pleura may occur in the later stages, either as a result of malignant infiltration, or of chronic infection.

DIAPHRAGMATIC PLEURISY

Diaphragmatic pleurisy may be a primary infection of the pleural membrane or secondary to disease of the lung or of the abdomen—for example, infections of the liver or gall-bladder or peritonitis from any cause.

During the acute stage the only radiological sign is, as a rule, impaired diaphragm movement on the affected side. Subsequently there may be found adhesions in the costophrenic or cardiophrenic angles, or small tent-shaped peaks which are often situated at

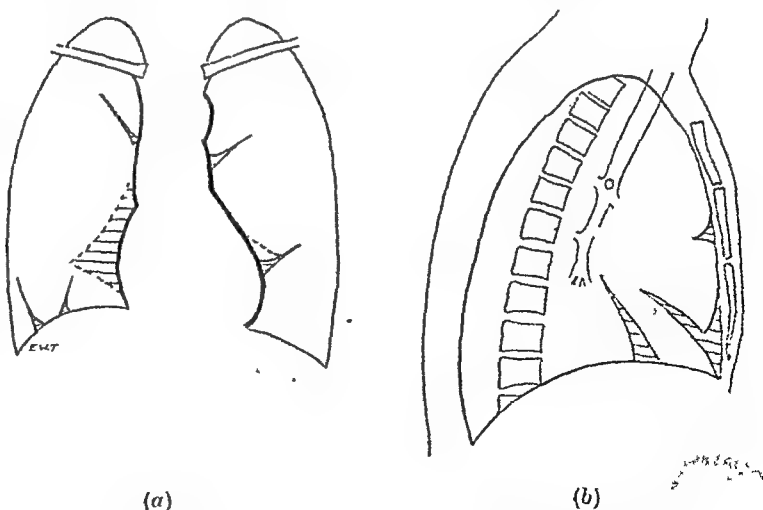


FIG. 282.—Diagram: "Peaks" on mediastinal and diaphragmatic surfaces resulting from old interlobar pleurisy.

the edge of the interlobar fissures, and are readily observed in the lateral view: from the peak of the triangle a fine line of sclerosis is often continued into the interlobar fissure. Such peaks and thickenings are often found at the conclusion of an attack of pneumonia or influenza, and in some cases there is chronic and rather intractable pain resulting from the diaphragmatic pleurisy. In one personally observed case the patient had recovered from an attack of pneumonia three months previously, but the main and horizontal interlobar fissures showed obvious sclerosis, and the patient complained of a constant dragging pain worse on deep breathing. Diaphragmatic pleurisy with or without effusion frequently accompanies a subphrenic abscess. The effusion tends to become localised and spreads across the lower chest in the form of a broad band parallel with the diaphragm. *Barjon* has described several such cases of localised diaphragmatic effusion. In the writer's own experience they have usually been associated with subphrenic abscess.

PLEURAL EFFUSION

Though this may result from various processes, infective or neoplastic, the radiological appearances are characteristic. It is never possible to say from the density of the X-ray shadow whether the effusion is serous, serofibrinous, hæmorrhagic, or purulent: a tendency to encapsulation always suggests the possibility of a purulent exudate, but is not conclusive. Hydrothorax may result from cardiac failure, or "back pressure," is usually bilateral and of limited extent: sometimes confined to the costo-phrenic angles.

Radiological Appearances

The radiological appearances depend mainly upon the amount of effusion present, upon the freedom of the lung to collapse, the presence or absence of adhesions, and the degree of atelectasis.

(a) Early effusions commence by filling up the posterior and lateral costo-phrenic angles. The margins of the lung are displaced therefrom, and the lower pole of the lung, contracting by its own resilience, takes on a rounded contour and floats on the effusion. The effusion spreads over the diaphragmatic surface, separating the lung from it, and, as a lateral film will show, spreads also up the posterior and, to a lesser height, the anterior chest wall, becoming gradually tapered off in an upward direction. In the postero-anterior view this tapering of the shadow along the axillary wall is easily recognised. The extreme upper limit of the layer of fluid cannot always be seen. *Kaunitz* has shown, by injecting light lipiodol, that the latter floats up to a level higher than the upper limit of the shadow, unless arrested by adhesions. The S-shaped curve mapped out upon the surface of the chest by percussion, which rises to its greatest height in the axilla (curve of Ellis), is not the curve which the radiogram shows at the upper border of the shadow of the effusion. This latter

curve is on the *deep surface* of the layer of effusion. It is sometimes due to the edge of the collapsed lung, and in thin effusions the lung detail can be traced down to the clearly defined lung edge. If the layer of effusion is thicker, the lung edge towards the base is lost in a hazy obscurity. The upper limit of the shadow is then formed by a summation of the shadows of the layers of fluid on anterior lateral and posterior chest walls, which becomes shallower in an upward direction. That this curved shadow can be so produced has been shown diagrammatically by *Fleischner*. Its upper limit is nearly always curved, with concavity upwards, and is higher on the lateral side than

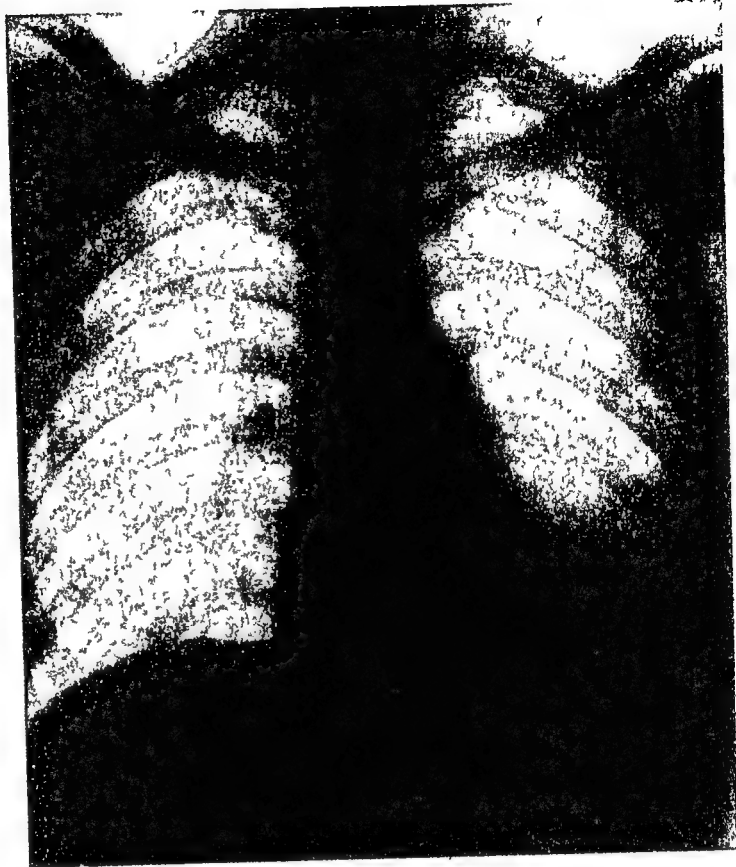


FIG. 283.—Left pleural effusion.

on the medial side. A similar but steeper curve is visible on anterior and posterior walls in the lateral view. Under certain circumstances this slope may be reversed. Occasionally the thin layer of fluid runs right up the chest wall, and may even cover the apex. The result is a marked loss of apical translucency, which may continue for some time after the absorption of the effusion. This narrow parietal shadow is more likely to occur when adhesions are present, limiting the collapse of the basal lung, but may occur in the absence of adhesions. It is not always composed entirely of fluid. A fibrinous deposit on the parietal, as well as on the visceral, pleura may be present. In cases of acute, as well as of chronic, pleuritis

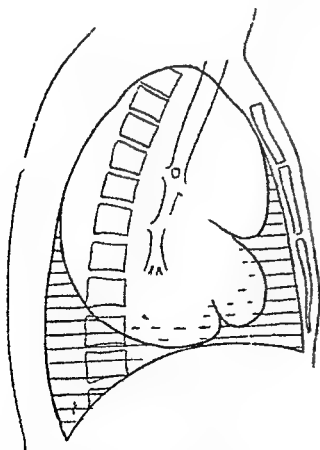


FIG. 284.—Pleural effusion, entering the fissures, lateral view.

treated by air replacement, by *Williamson*, and studied radiologically by the writer, this fibrinous layer was observed adhering to the parietes after the lung had collapsed, and showed the imprint of the lobes, with a small peak at a point corresponding with the edge of the middle interlobar fissure. The line of pleural thickening is also commonly observed after operation for empyema, while the lung is collapsed, and it may be observed at an early stage in the development of empyema, before any considerable effusion has occurred. It is, therefore, a valuable diagnostic sign, especially in children. In very large effusions the whole hemithorax is opaque, though usually a slight translucency persists at the inner part of the extreme apex.



FIG. 285 —Left pleural effusion, running up axillary wall.

Reversal of the Normal Curve. — The normal slope of the "effusion curve," upwards and outwards, may be reversed under the following conditions:

(1) When limited to the basal region by adhesions (diaphragmatic effusion) In such cases the effusion is usually purulent.

(2) When atelectasis of a lobe is present. This has been pointed out by *Westermarck*, who

found this reversal of the line of pleural effusion in eighteen cases of atelectasis, usually of the lower lobe, sometimes of the middle and upper lobes. In eleven cases the atelectasis was caused by bronchial carcinoma. The bulk of the effusion usually lay near the area of atelectasis, and since the medial parts of the lung were in most cases more retracted, the effusion tended to collect towards the middle line. In the supine position it spread out over the whole hemithorax. The frequent association of this sign with bronchial carcinoma should be noted.

(3) During reabsorption of the fluid. This is not a common finding. As a rule, during absorption, the curve maintains its characteristic shape, but gradually retraces its steps. Sometimes the slope is reversed (downwards and outwards) during reabsorption. It seems probable, in view of *Westermarck's* findings, that this may be explained by incomplete re-expansion of the lower lobe ("relative atelectasis").

Effect of Effusion

ON THE MEDIASTINUM this is almost invariably displaced to the opposite side from the first. With large effusions the displacement is most marked. This displacement is a valuable differential sign in cases showing unilateral obscuration. There is but one other condition which shows the same finding, namely, infiltration of the whole lung by diffuse alveolar carcinoma (*Sante*). If there is displacement towards the opaque side the cause is usually atelectasis (massive collapse), fibrosis or growth with atelectasis. If there is no displacement, pneumonic consolidation, or atelectasis or growth, combined with a moderate amount of effusion, may be present.

ON THE RIBS.—With large effusions the chest wall may bulge on the affected side, and the ribs run more horizontally, with widened intercostal spaces. This can also occur in the massive form of carcinoma just mentioned. Only the exploring needle can decide the issue.

ON THE DIAPHRAGM.—This shows absent movement for the first. In large effusions the diaphragm is depressed.

Behaviour of Effusion after Tapping.—After tapping, even after the withdrawal of large amounts of fluid, the level of the effusion and even the degree of opacity are usually little affected. This is because the chest wall and mediastinum revert to their original position and the lung expands slightly. The effusion—though diminished in quantity—has, therefore, a smaller space to occupy, and its level may be quite unaltered. Tapping, with the intention of uncovering a suspected lesion in the lung, is usually disappointing. Air replacement is usually more successful, but may also fail if the lung is much collapsed: contrast between the collapsed lung and diseased lung may then be insufficient for diagnosis. Traces of air often enter the chest accidentally during paracentesis and show a fluid-level at the top of the effusion. Occasionally the air is caught in a pocket formed by adhesions, and then shows as a bubble with fluid-level in the midst of the opaque area. Its superficial situation and transient existence distinguish it from an abscess or cavity in the lung.

Effect of Posture in Pleural Effusion.—As a rule, little if any displacement of an effusion is noted if the patient is examined lying down. Such displacement is more readily shown if the patient is examined while lying on the affected side. If atelectasis is present, the shadow shift in this position is more marked.

Penetration into Interlobar Fissures.—Unless the margins of the interlobar fissures are sealed by adhesions, the effusion runs into these. It is not un-

common to see a dense line of fluid running along the middle interlobar fissure. Still more commonly does the lower end of the main fissure gape and become filled with effusion. The lateral view shows the characteristic appearance of an effusion running into all the interlobar fissures, and the consequent rounding of the sharp margins of the lobes (Fig. 284). A parieto-interlobar effusion at the lower end of the main fissure may be simulated by the shadow of the heart and inferior vena cava overlapping that of the diaphragm. The distinction by screening is easy.

ENCAPSULATED PLEURAL EFFUSIONS

These may be :

- (1) Parietal.
- (2) Diaphragmatic.
- (3) Mediastinal.
- (4) Mediastino-interlobar.
- (5) Interlobar.

Encapsulated pleural effusions commonly result from a pneumonic infection, and are usually purulent. It is, however, well known that serous encapsulated effusions may occur, and there are no means of distinguishing them radiologically except by their course.

Parietal Encysted Effusions

These show certain sites of election : (1) along the lateral wall ; (2) along the posterior wall, usually near the base. They may occur very rarely at the apex or on the anterior wall. Decubitus seems to favour the frequent occurrence on lateral and posterior walls. Special mention must be made of the small encysted effusion which so often occurs in the upper part of the lateral chest wall. Its greatest depth may lie at a point high up in the axilla, and such an effusion may be missed by percussion or by the exploring needle unless it is realised that it lies opposite the second or third interspace in the axillary line. The exact position should be mapped out by the radiologist, using tangential projection.

Parietal encysted pleural effusions show a well-defined border convex towards the lung. The lung shadows are displaced. Occasionally the encysted effusion has a less definitely rounded edge, but is flatter and extends some distance round the chest wall. An endeavour must always be made to find by tangential views the point of maximum depth of such effusions, usually situated near the middle of the shadowed area. This point should be selected for paracentesis ; elsewhere the needle may pass through the thin layer of fluid and fail to withdraw any of it.

Occasionally a parietal effusion is situated at the edge of an interlobar fissure, and penetrates for some distance into it. The shadow is then

somewhat wedge-shaped, and continuous at its apex with the interlobar fissure as a line of sclerosis. In one situation this is easily mistaken for a consolidation of the lung, namely at the upper posterior end of the oblique fissure. In the lateral view the triangular shape of the "parieto-interlobar" exudate is not unlike that of a consolidation of the apex of the lower lobe. As a rule, the latter has an ill-defined lower margin, while the effusion is sharply defined above

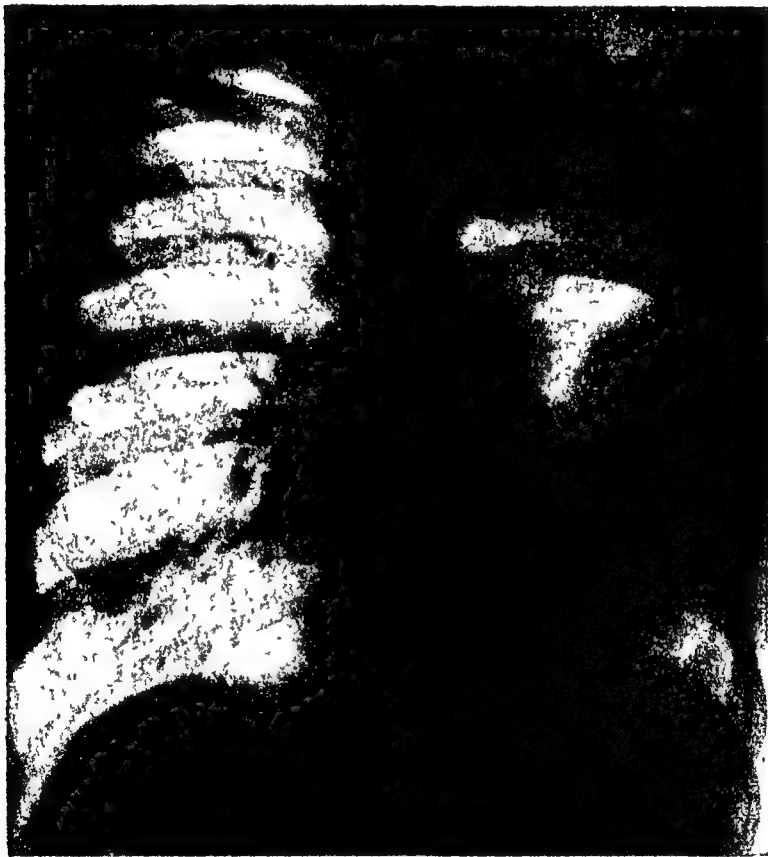


FIG. 286.—Multiple encysted parietal effusions. Spontaneous reabsorption of the fluid occurred later.

and below. The occasional occurrence of an accessory lobe at the apex of the lower lobe (*Pohl*), which, when consolidated, is sharply defined both above and below, adds to the difficulty in diagnosis. In such a case reliance must be placed in the character of the edge (convex in effusion, straight or concave in consolidation). Rupture into a bronchus, leading to spontaneous cure, is not uncommon. When it occurs, air and a fluid-level are visible within the opaque area. The fluid-level may be hidden behind the diaphragmatic cupola in the postero-anterior view and be projected on to the spine in the lateral view, where

it may be confused with the margin of a vertebral body. An antero-posterior radiogram, with the patient leaning forward and tube raised, will bring it above the diaphragm. A lateral view in the same posture will differentiate the fluid-level from vertebral bodies. Sometimes parietal encysted effusions are multiple; there may be two close together, as in Fig. 286. The writer has seen a few cases in which parietal effusions and mediastinal encysted effusions have co-existed. The differential diagnosis is usually from growth. When basally situated a rounded well-defined shadow is shown in the postero-anterior view. The lateral or tangential view determines the parietal situation. This does not, however, exclude neoplasm, since secondary pleural deposits have much the same shape, and even primary lung tumours sometimes lie on the pleural surface, in contact with the ribs. Elevation and fixation of the adjacent part of the diaphragm with obliteration of the costo-phrenic cul-de-sac points to an inflammatory lesion, and is in favour of a basal parietal encysted effusion.

Differential Diagnosis.—Endothelioma, hydatid cyst, subpleural fibroma, tubercular abscess (rib or spine), sarcoma of rib, and subpleural hæmatoma are other conditions which enter into the differential diagnosis. Very careful search must be made for evidence of rib involvement showing inflammatory or neoplastic characteristics: usually the history and clinical signs are decisive.

Mediastinal Encysted Effusions

These develop between mediastinal and visceral pleura. They are not always confined to the mediastinal surface of the lung, but may extend back-

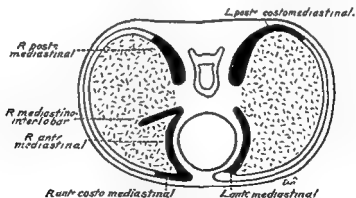


FIG 287.—Diagram Sites at which mediastinal effusions tend to become encysted (After Engel and Schall)

wards round the lung edge (see Fig. 287). The root of the lung and the ligamentum latum divide the mediastinal surface vertically into two compartments. A mediastinal encysted effusion may occur either in the anterior or posterior compartment, or in both. The writer has seen a case which presented two convex shadows along the right heart border, one of which was shown by rotation of the patient to be in front of the hilum, the other behind. In the

upper part of the anterior shadow was an air bubble with fluid-level. The diagnosis of combined anterior and posterior mediastinal effusion was made (Fig. 288). Within a week the anterior effusion was entirely coughed up. The posterior effusion also cleared up in the same way, and the patient made a



FIG. 288.—Encysted mediastinal effusion. Anterior and posterior mediastinal effusions on right side. The anterior effusion communicates with a bronchus, and shows a fluid-level. Both were spontaneously evacuated and the chest became normal.

good recovery. A few months later the lung fields were normal, except for light thickening around the hilar shadows.

A posterior costo-mediastinal effusion lying in the paravertebral region may fail to show clearly in a full lateral view, since its edge is obliquely opposed to the ray and not tangential to it. Thus in the case illustrated in Fig. 289, of right

costo-mediastinal empyema, the effusion showed only a faint shadow in the right lateral view, and was still more ill-defined in the right anterior oblique, in which the rays passed through the shortest diameter of the encysted fluid. But in the left anterior oblique view the typical lens shape of the effusion, with its curved, well-defined pulmonary border, was clearly seen. In such a case the right posterior oblique view would have been equally suitable.

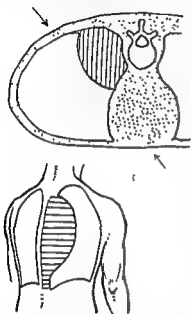


FIG 289.—Diagram: A posterior mediastinal encysted effusion lying paravertebrally may fail to show a well-defined edge in lateral projection, an oblique view (arrows) will show the edge distinctly

other views are sometimes useful, especially the hollow-back lordotic position and oblique views.

Shape of Interlobar Effusions.—The general form might be roughly imitated by placing two soup plates face to face—the rims of the plates representing the zone of marginal sclerosis and adhesion in the interlobar fissure which seals off the effusion. This biconvex form, seen edge-on, gives a spindle-shaped shadow, with short lines of sclerosis at the ends of the spindle. If the effusion is tense, the spindle becomes broader, or nearly circular. Sometimes the lenticular form is absent, the effusion being spread out in a sheet along an interlobar fissure. The spindle or linear shadow is in any case only visible when the effusion lies edge-on to the rays, and it is necessary to consider the various appearances according to the situation of the effusion.

Engel has described, in children, an anterior superior mediastinal effusion, usually pneumonic in origin, more common on the right side, limited towards the lung field by a sharp vertical border, and showing on the right side a projecting angle at the level of the horizontal fissure. The latter feature distinguishes it from an enlarged thymus.

In regard to the differential diagnosis of mediastinal effusion, *Van der Mandele* mentions dilated left auricle, retrocardial aneurysm, cold abscess of spine, spondylitis deformans, paracæsoophageal hernia, dilated œsophagus (cardiospasm), scoliosis, pericardial fat. The differential diagnosis should present no particular difficulty. To this list might be added pericardial diverticulum, ganglioneuroma of vagus (*Assmann*), and new growth of the pericardium (own observation).

Interlobar Encysted Effusions

Interlobar pleural effusions may be limited to one part, or extend from top to bottom of an interlobar fissure. A lateral view is essential to their detection and localisation, but

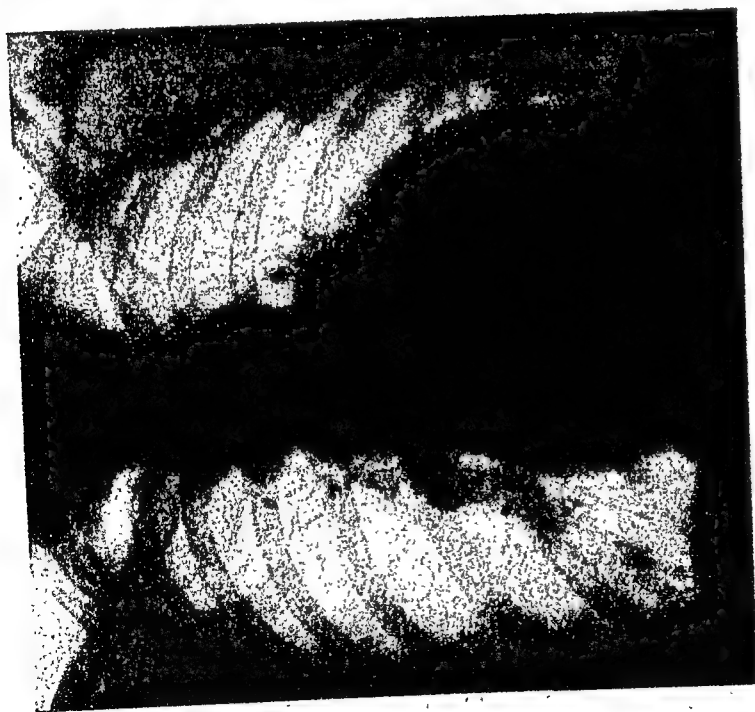


FIG. 290.—Mediastinal effusion. The left border of the heart is obscured by a well-defined shadow with convex margin, due to fluid encysted between mediastinal and visceral pleura. No visible pulsation. Operation: recovery.



FIG. 291.—Interlobar effusion in the right middle fissure.

(1) **IN THE MIDDLE INTERLOBE.**—Here the effusion lies nearly horizontally, and is edge-on to the rays in the postero-anterior view. The spindle-shaped shadow may be seen, usually in the *outer* part of the middle lung field, because the horizontal interlobe rarely reaches the mediastinal surface of the lung, and is often shallow.

The middle interlobar fissure often has a slight dip downwards and forwards. A slight elevation of the tube, or backward tilt of the patient, will be needful to bring its plane edge on to the ray.

The lateral view shows a band of shadow in the middle of the anterior half



FIG. 292.—Pneumothorax interlobar empyema. Present four years. Operation recovery.

of the lung, extending from the hilum to the anterior chest wall, above the middle lobe.

(2) **IN THE MAIN FISSURE.**—The effusion in this case necessarily lies obliquely, and in the postero-anterior view is traversed by the rays, *not* from edge to edge, but from face to face. The relatively slight amount of fluid interposed to the rays, in this view, may cause the shadow to be faint; moreover, owing to the tapering off which occurs in its edges, these are sometimes ill-defined, particularly the upper edge, since the bulk of the fluid tends to gravitate downwards. For this reason the lower edge is usually better defined than the upper (Fig. 293). If the effusion is not localised, but extends through the whole length of the main interlobe, the appearance is that of a diffuse opacity of the hemithorax, limited below to the outer side by a well-defined line (edge of

the main fissure). Sometimes it extends beyond the edge of the fissure into the main pleural cavity, filling up the costo-phrenic angle.

The most usual shape of the effusion, in the postero-anterior view, is that of a hatchet-shaped opacity in the middle part of the lung, with ill-defined or moderately well-defined edges and narrower towards the hilum; a triangular region of translucent lung usually separates it from the heart shadow and diaphragm, and the costo-phrenic angle is clear. The reason for the translucent area so often seen in the right cardio-phrenic angle is not clear. In some cases it may be explained by the anatomical fusion of middle and lower lobes, shown by *Kreuzfuchs* and *Schumacher* to occur at this site. In other cases it must be assumed that the edge of the effusion has been sealed off here by adhesions of the interlobar surfaces. This typical form is not always found: the shadow may be oval, with long axis vertical, or irregular in shape.

The upper part of the oblique fissure on the right side is more horizontal than the lower half. Effusions in the upper part of this fissure may therefore show broad bands of shadow, or even spindle-shaped or circular shadows in the postero-anterior view. They are, as a rule, separate from the hilum and mediastinal shadow, but may extend inwards to fuse with it.

The writer once saw a rounded opacity projecting from the right mediastinum, with a well-defined hemispherical contour, just above the hilum. The chronic history and age of the patient were in favour of mediastinal tumour. Autopsy showed a small localised interlobar empyema in the upper part of the main interlobe close to the mediastinal edge. There was no evidence of neoplasm.

It follows from the usually flattened form of interlobar effusions, and from the anatomy of the interlobar fissures, that in the postero-anterior view illumination of the plane of the fissure will intensify the shadow, and show the clear-cut margins so typical of this condition.



FIG. 293.—Chronic interlobar empyema showing slight displacement of the œsophagus, simulating mediastinal tumour or cyst. Same case as Fig. 292.

Postero-anterior Position.—For the illumination of the oblique interlobe in the postero-anterior view, we may follow the rule given by *Beclère* and *Holz-knecht*: namely, in the postero-anterior projection, raise the tube and lower the eye; in the antero-posterior projection, lower the tube and raise the eye, viewing it from above and behind. With most modern screening stands the range of movement of the apparatus is insufficient to allow of this, and a backward or forward inclination of the patient's body, as the case may be, must be used to supplement it. The lordotic or hollow-back position (*Fleischner*), illustrated in Figs. 299(b) and 301, is an extremely useful addition to the technique, since it enables one to demonstrate more clearly the oblique interlobe on both sides of the chest throughout its entire extent. The patient is instructed to lean backwards with head and shoulders resting on the back

support and arching the back, to protrude the abdomen. The oblique interlobe thus becomes horizontal and can be illuminated along its length. This is in many cases the only suitable method for showing the nature and extent of mediastino-interlobar effusions, and is very easily carried out in children, in whom this form of effusion is common. A mediastino-interlobar effusion shows typical appearances described later: sclerosis of the interlobar pleura of the main fissure shows an oblique line crossing the chest, usually convex upwards. The spine of the scapula also crosses the lung field in this position, and must not be mistaken for an interlobar shadow.

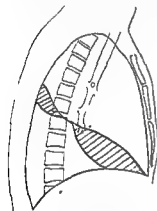


FIG. 294.—Interlobar effusion in main fissure, lateral view

Lateral Position.—It is, however, to the lateral position that we must turn for the most satisfactory evidence in the localisation of interlobar effusions, just as it is the most useful in the localisation and study of lobar lesions. In the lateral position, these effusions, whether in the main or horizontal interlobe, are nearly edge-on to the rays.

Interlobar effusions may occur in two ways:

- (1) By limited infection arising in or near an interlobe and becoming shut off in this situation.
- (2) By penetration into the interlobe of a general effusion with subsequent encystment in the interlobe. This form is probably rare.

Radiological Signs of Encysted Interlobar Effusion

- (1) They are usually of a lenticular shape, flattened from above downwards, giving, in the lateral view, spindle-shaped shadows, the long axis of which is in the line of the interlobe affected.
- (2) Both borders of the shadow are clearly defined against the lung. This

is a most important sign, and is due to the limitation on all sides by a layer of interlobar pleura. For various reasons, such as the curvature or twisting of the interlobar spaces, or of the overlapping by spine and heart shadows, we cannot always show both borders sharply defined in the one and same position, but by slight changes in position first one border and then another is shown to be clearly defined. Interlobar effusions do not cause atelectasis of the immediately adjacent lung.

If, however, one edge is ill-defined, the cause of this is in the adjacent lung disease, and may be either infective or neoplastic. Either condition may spread to the adjacent interlobe and cause an effusion. An interlobar empyema occurring before resolution of the pneumonia which has caused it may, therefore, show an ill-defined shadow on the side adjacent to the consolidated lung.

(3) The small peaks or thickenings of the interlobe given off from the edge of the interlobar effusion and continuing into the interlobe have already been referred to. They are an exceeding common and typical finding.



FIG. 295.—Interlobar effusions in all fissures, right side.

Bearing these facts and the anatomical relation of the interlobes in mind, a diagnosis of interlobar empyema is, as a rule, not difficult. They are, however, occasionally overlooked, with serious results. The case illustrated in Fig. 293 is that of a girl, aged 18, who had been four years in a sanatorium with a diagnosis of phthisis. The appearances in the postero-anterior film at the end of that period were those of a rounded, nearly circular, shadow, very

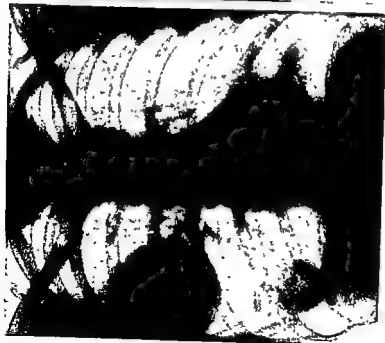


FIG 296 Old calcified tuberculous lesion at right base and calcified interlobar empyema in right main fissure F 39 No chest symptoms Tuberculous adenitis present in right axilla.



Lateral view. The interlobar effusion is anteriorly with the sclerosed horizontal fissure

clearly defined below and fading off above. In the lateral view this shadow, again of circular outline, was in the position of the upper part of the main interlobe and showed typical marginal peak. At operation the surgeon found a pneumococcal interlobar empyema, which was drained, and the patient made a good recovery.¹ *Fleischner* has published a case showing almost exactly similar appearances, due to hydatid cyst in the interlobar fissure.

Interlobar effusions in the oblique fissure may extend throughout the whole fissure or be limited to the upper or lower half. Those in the lower half often show a roughly triangular outline in the lateral view, with the apex at the hilum level, the axis directed obliquely downwards and forwards. The effusion may be tapered at both ends or may broaden out towards the anterior end. In the latter case, differential diagnosis from a middle lobe-consolidation is by no means easy. Help may be afforded by observation of the lung shadows belonging to the middle lobe, which lie above an effusion but are included in a consolidation. A consolidated middle lobe is often atelectatic as well, which increases its resemblance to an interlobar effusion. In such cases it may be possible to observe clear streaks within the shadow, due to translucent bronchi, contrasted with the surrounding atelectatic and consolidated lung. This appearance is never seen in an effusion. If the shadow of an effusion is crossed by lung shadows, these appear as dense streaks (due to vessels) intensified by the background of the shadow of the effusion. This point is often very helpful in the differential diagnosis between consolidation and effusion in any part of the lung. In some cases it is necessary to use lipiodol to make the distinction. If the consolidation of the middle lobe is only partial, the diagnosis is usually much easier, since in one view or other the shadow will show in some part a fuzzy ill-defined edge. It should be remembered that interlobar empyema is a somewhat rare condition—middle lobe consolidation or atelectasis is much more common.

Method of Resolution of Serous Interlobar Effusions

These may (1) require surgery, or (2) be coughed up, or (3) may be slowly absorbed. In the last case, the clearly defined margins are retained, and the shadow gradually shrinks in size. This appearance is quite different from the resolution of a consolidation, which shows gradual clearing of the middle of the shadow, as the lung parenchyma becomes aerated, though the bounding line of the interlobar sclerosis often persists during and after complete resolution. If atelectasis has accompanied the consolidation, the lobar shadow becomes *larger* and gradually more translucent as the atelectasis clears up, while an absorbing effusion becomes *smaller*, but remains homogeneous. Repeated observations, therefore, will make the diagnosis clear in cases in which a single observation has failed to do so.

¹*Prof. E. D. Telford*, personal communication.

MINOR INTERLOBAR PLEURISIES

The radiologist is constantly meeting with minor interlobar pleurисies, often residual from previous disease, which may take the form of :

(1) Limiting interlobar sclerosis, combined with disease of a neighbouring lobe or lobes.

(2) Linear thickenings in an interlobe, without evidence of pulmonary disease.

(3) Encysted serous effusions, completely shut off in a part of an interlobar space. These are rather rare.

(4) Combined interlobar or peripulmonary pleural thickening, taking the form of triangular peaks at the surface of the lung, and often prolonged into the interlobe as a line of thickening. A localised parietal thickening often accompanies them. These are common.

(5) Mediastino-interlobar pleurисies. These have only recently been studied in detail. They are extremely common, but unless sought for deliberately by a special technique, they are overlooked.

Linear Interlobar Sclerosis, either occurring alone or combined with a tuberculous, malignant, or other lesion of the lung, is familiar to radiologists. It is sometimes assumed that the horizontal interlobe is a site of election because it alone shows in the ordinary postero-anterior film, but the routine use of the lateral view shows that this is not the case, and, as a rule, when the horizontal interlobe is thickened, the oblique interlobe is affected as well. Often the oblique interlobe is affected alone. Tuberculosis sometimes spreads along the interlobe ("scissural type" of *Barjon*), and a band of infiltration in the upper lobe sharply limited below by the horizontal interlobe is a well-known radiological picture. Tuberculous infiltrations along the oblique interlobe can be demonstrated in many cases, usually in the upper lobe. When a large part of a lobe is affected, the sharp limitation of the disease at the oblique interlobe is readily shown in the lateral view. Growths sometimes show a similar limitation.

Pneumonia shows a distinct tendency, during resolution, to linger in the region of the interlobe, so that a case of delayed resolution should always be studied with special reference to the interlobes, making full use of the lateral and hollow-back position. In this way it is possible to study progress of resolution in a manner and with a degree of accuracy unattainable by the ordinary postero-anterior film. After resolution some thickening of the interlobar pleura often persists for many months.

Parieto-interlobar Effusions.—These are interlobar pleurисies occurring near the surface of the lung and due to a combination of peripulmonary and interlobar pleurisy. They therefore occur at the edges of the interlobar clefts. They are characterised by small triangular opacities on the lung margin with their pieces prolonged into an interlobe, and they usually show slight rounding of the

lung margins. In these cases there is fluid, or possibly fibrin or organised fibrous tissue, in the triangular space. The parietal part of the thickening may show as a faint line up and down the chest, but is often invisible. Their common sites are indicated in Fig. 283. These triangles can arise in two ways. Firstly, as a result of a general pleural effusion; secondly, from a limited pleurisy. If we watch the development of a large pleural effusion, we sometimes see it run into an interlobe. The lung apparently collapses, not all in one piece, but lobe by

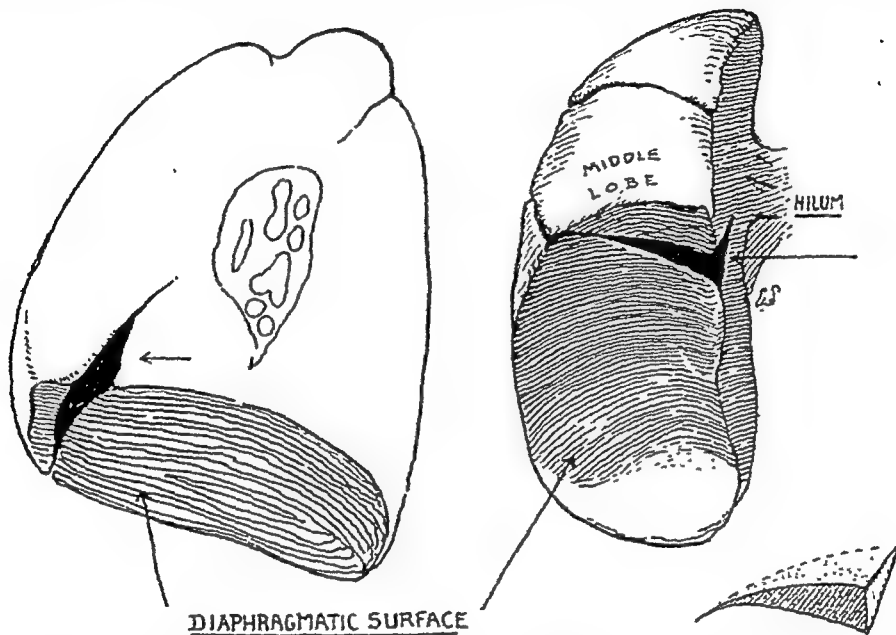


FIG. 297.—Diagram: Mediastino-interlobar effusion. This forms a thin, flattened layer, tapering to an edge posteriorly and externally, in the lower end of the main fissure.

lobe, especially in children, in whom adhesions between the lobes are rare, and the effusion then penetrates between the lobes.

Parieto-interlobar pleurisies may account for many of the common diaphragmatic "peaks" or "tents." A parieto-interlobar effusion or thickening extending from the diaphragm into an interlobe would in many cases afford a more satisfactory explanation for these "peaks" than does the theory that they are due to simple adhesions or to retraction and dimpling of the under-surface of the lung.

The writer has seen several cases in which angular diaphragmatic tents were seen in the lateral view to be continuous at their apices with a thickened main interlobe. As we shall have occasion to see when discussing mediastino-interlobar pleurisy, a rather similar explanation holds good for pericardial adhesions which may be seen projecting from the left or right heart border.

Mediastino-interlobar Pleurisy.—This group is particularly interesting on account of the great frequency, especially in children, and of the extreme

characteristic appearances found on X-ray examination when they are carefully investigated. The first observations which *Fleischner* reported in 1924 seem to have passed unnoticed in this country for a long time. The condition is found in children (rarely in adults) who usually give a long history of chronic chest trouble, often extending back to infancy. They may show visible hilar

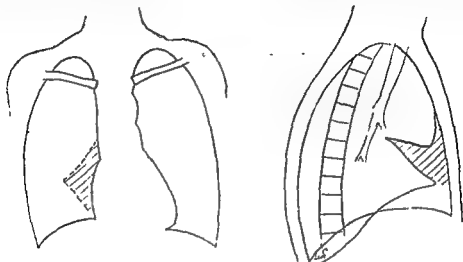


FIG. 298 —Diagram. Mediastino-interlobar effusion, in anterior and lateral projection.



FIG. 299 —(a) Diagram. Mediastino interlobar effusion in lordotic position.



(b) Lordotic (hollow-back) position.

gland enlargement, but not always. The condition is an effusion or a fibrinous exudate occupying the lower end of the oblique fissure in its median half. Common in children, it has been attributed by *Fleischner* to infection of this interlobar space by a tuberculous lymphatic gland which lies in the lower hilum, separated from the fissure only by a layer of pleura. Symptoms are often slight or non-existent. The writer examined 100 children *seriatim* from

the waiting-hall of an Outpatient Department, and found six cases of this condition. Most had suffered from the usual exanthemata, bronchitis, and whooping-cough, but none showed evidence of active pulmonary tuberculosis.

The effusion or fibrinous layer shows, in the antero-posterior view, a barely perceptible shadow, which becomes intensified as the child leans backwards

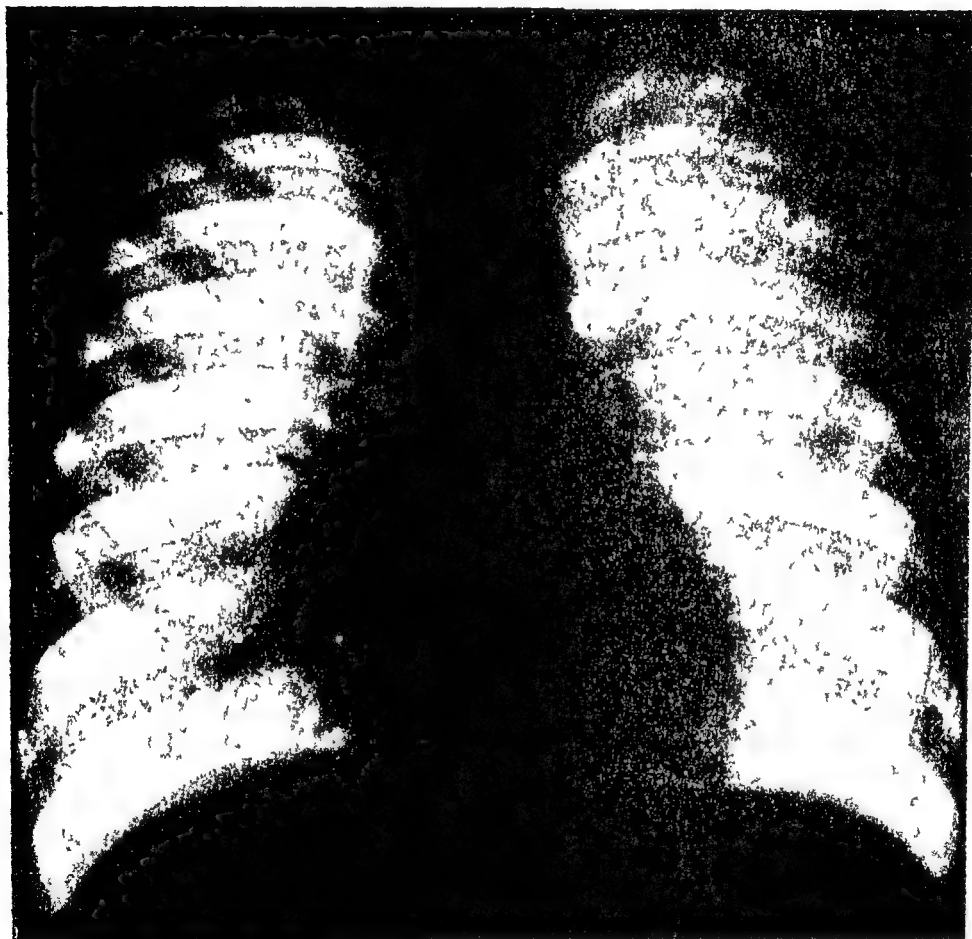


FIG. 300 —Mediastino-interlobar effusion. Erect position. Note shadow extending downwards from the right root For same case in lordotic position see Fig. 301.

towards the tube until the complete hollow-back position is reached, when the main interlobar fissure is horizontal and directly in the line of the rays (Fig. 299). Before the hollow-back position is fully attained, it is evident that the apex of the triangle is directed outwards and lies posteriorly. Below this the lower border gradually approaches the mediastinum. In the full hollow-back position a characteristic triangle is seen, with apex outwards, continued outwards as a line of interlobar sclerosis, convex upwards. Careful examination shows that there are often two triangles, a short anterior one and a longer,

posterior one. Their apices are connected by the linear thickening in the interlobe. Both triangles have their bases in contact with the mediastinal shadow. The smaller anterior triangle is the surface of contact between the anterior end of the effusion and the anterior chest wall or diaphragm. The larger posterior triangle is the sheet of effusion or fibrinous exudate extending outwards into the interlobar fissure, sealed off externally by a fusion of the



FIG. 301.—Mediastino interlobar effusion, lordotic position

interlobar pleura between lower and middle lobes. The shape and position of the effusion are shown in the diagram (Fig. 297). Radiologically the lateral view brings its situation out very clearly. It is shown as a triangle, apex behind at the hilum level, at the junction of horizontal and oblique fissures, base anteriorly in contact with chest wall or diaphragm. The whole shadow is usually convex upwards.

It is a moot question whether the "double triangle" occurs in any other condition. Some observers believe that it may occur in consolidation with



FIG. 302.—Mediastino-interlobar offusion, lower end of main fissure. Right lateral view.



FIG. 303.—Mediastino-interlobar pleurisy. Right lateral view.

atelectasis of the middle lobe. This is unlikely. The middle lobe is pyramidal, with a broad anterior surface. When it is atelectatic, this broad anterior surface can hardly become *smaller* than the rest of the lobe, and could not be contained in the small anterior triangle. This anterior triangle is obviously identifiable with the lower anterior blunt end of the mediastino-interlobar effusion, to which the outer border is seen to converge.

A further point of differentiation lies in the observation of the lung details in the lateral and antero-posterior views. If the vascular trunks to the middle lobe can be recognised in either view, not included in the shadow, but lying

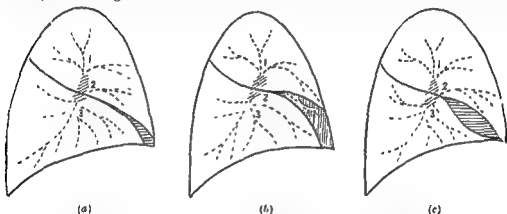


FIG. 304.—Diagram Right lateral view. The pulmonary vessel shadows in relation to (a) mediastino-interlobar effusion, (b) middle lobe consolidation or collapse, (c) effusion in lower end of main fissure.

above it, the shadow must be interlobar, and cannot be a collapsed or a consolidated middle lobe (Fig. 304).

Usually present on the right side, a mediastino-interlobar effusion may occur on the left side also. The writer has seen one appear on the right, clear up in a few weeks, then appear on the left, and again clear up rapidly. A pleuroparietal adhesion may persist as a sequel of this condition. Occasionally it is observed in malignant disease as part of an early malignant pleurisy.

DIFFERENTIAL DIAGNOSIS OF MEDIASTINO-INTERLOBAR PLEURISY.—There are eight conditions in which a triangular shadow is seen bordering the heart, with its apex above, its base on the diaphragm. These are :

- (1) Mediastino-interlobar pleurisy (effusion or sclerosis). Antero-median.
- (2) Costo-mediastinal pleurisy, posterior or anterior.
- (3) Atelectasis of lower lobe. Postero-median.
- (4) Atelectasis or consolidation of accessory lobe (Gräberger). Median.
- (5) Consolidation with atelectasis of midlobe. Antero-median.
- (6) Elevation of central part of diaphragm. Median.
- (7) Mediastino-diaphragmatic pleurisy. Median.
- (8) Pericardial fat.

CHAPTER XXXIV

TUMOURS AND CALCIFICATION OF THE PLEURA

TUMOURS OF THE PLEURA

Benign primary tumours of the pleura are rare. They include fibroma, fibro-endothelioma, angioma, chondroma, osteoma, and simple cyst.

Of the *malignant primary tumours* the following may occur : endothelioma, primary sarcoma, fibrosarcoma, and chondrosarcoma.

Tumours of the pleura show rounded shadows, sharply defined, usually attached to the parietes by a broad base. In tangential projection the pleural membrane may be seen passing from the side of the tumour into the chest wall. Artificial pneumothorax is often necessary for diagnosis.

Sarcoma occurs in three forms : (1) Diffusely infiltrating, which cannot be distinguished from endothelioma : (2) circumscribed, resembling an encysted effusion but found to be solid on puncture, and, as a rule, remaining on the chest wall after diagnostic pneumothorax. This type sometimes, however, invades the lung. Reduction in size of the tumour may follow X-ray treatment. (3) A rare form, the giant pleural tumour, of which *Lenk* collected eight cases, lies almost free in the pleural cavity, usually arises from the diaphragmatic pleura, and does not invade the lung. Effusion is rare in this form.

Endothelioma (mesothelioma) occurs in the pleural sac, usually near the base of the lung, as a flat or nodular mass, involving both parietal and visceral pleura. Nodules may occur in mediastinal lymph nodes and ribs, and distant metastases in other organs. The growth may completely envelop the lung. Serous or blood-stained effusion occurs, which tends to be "pocketed" by fibrosis.

Willis believes that the great majority of all reported cases of "primary" endothelioma of serous membranes are secondary to primary tumours of neighbouring viscera, and quotes *Robertson* to the effect that "No diagnosis of primary carcinoma, mesendothelioma or endothelioma of the pleura can be justified on any logical ground."

Erwing, however, believes that primary endothelioma of serous membranes is highly characteristic.

According to *Osler*, primary endothelioma of the pleura is not uncommon, *Keilty* being quoted as having found nine cases in 5,000 autopsies. Prominent symptoms are pleuritic pain, cough, friction, progressive effusion, and dyspnoea. The effusions are usually blood-stained, but may be clear at first. There is usually progressive weakness, with emaciation and anæmia.

DIAGNOSIS.—These cases are likely to present themselves radiologically:

(a) As a pleural effusion, rapidly recurring; nodulation may be visible if the effusion is not too complete.

(b) As a pleural thickening, with or without nodules projecting from the parietes into the lung fields, usually with well-defined contours.

(c) As a large mass, simulating effusion, but solid on puncture (end stage of case).

(d) As a shadow resembling diaphragmatic effusion—in the case of an endothelioma arising on the diaphragmatic surface.

(e) As a mediastinal tumour, projecting into the lung field and possibly occluding the bronchi.

Artificial pneumothorax may clear up the diagnosis.

The radiological and pathological appearances are illustrated by a case (Figs. 303 and 306) for which the writer is indebted to Dr. Beath and Prof. Young.

The details of this case are as follows:

History.—Previous history good. One year ago pleuritic pain right chest for one week. Two months ago pain in chest recurred. Dyspnoea. Sign of pleural effusion. No sign of primary malignancy elsewhere. Effusion tapped five times, rapidly recurred. Ten pints removed in all. Losing weight.

X-ray Appearances.—Right pleural effusion, with suspicion of underlying infiltration of lung.

Two months later, the pleurisy recurred. After artificial pneumothorax multiple round shadows on parietal pleura became visible, clear of lung, and suggesting pleural endothelioma. The patient died six months later. Autopsy showed the right pleural sac filled with a friable necrotic tumour.

Prof. Young has kindly sent the following description:

"The lung is ensheathed by a layer of cellular tumour tissue. On the parietal surface the growth has extended into the interlobar fissure, and on the diaphragmatic surface it thickens to form a larger mass. The mediastinal surface presents two large homogeneous masses of growth, which are probably glandular. The lung is compressed and not grossly invaded by the tumour.

"Microscopically the structure is alveolar, frequently associated with a papillary proliferation of the tumour cells. The latter are spheroidal or cuboidal, and contain hyperchromatic vesicular nuclei with two nucleoli. The periphery of the lung shows a plasma cell infiltration and is, as a rule, sharply demarcated from the growth by a layer of vascular connective tissue. This layer of connective tissue apparently represents the deeper part of a thickened visceral pleura and is only occasionally penetrated by the growth.

"The mediastinal nodules are probably glandular. They contain no surviving glandular structure, but their location is most suggestive."

Simple Cysts of the Pleura.—These are rare and of doubtful ætiology. Sixteen cases are on record. *Freedman* and *Simon* have described a cyst of the

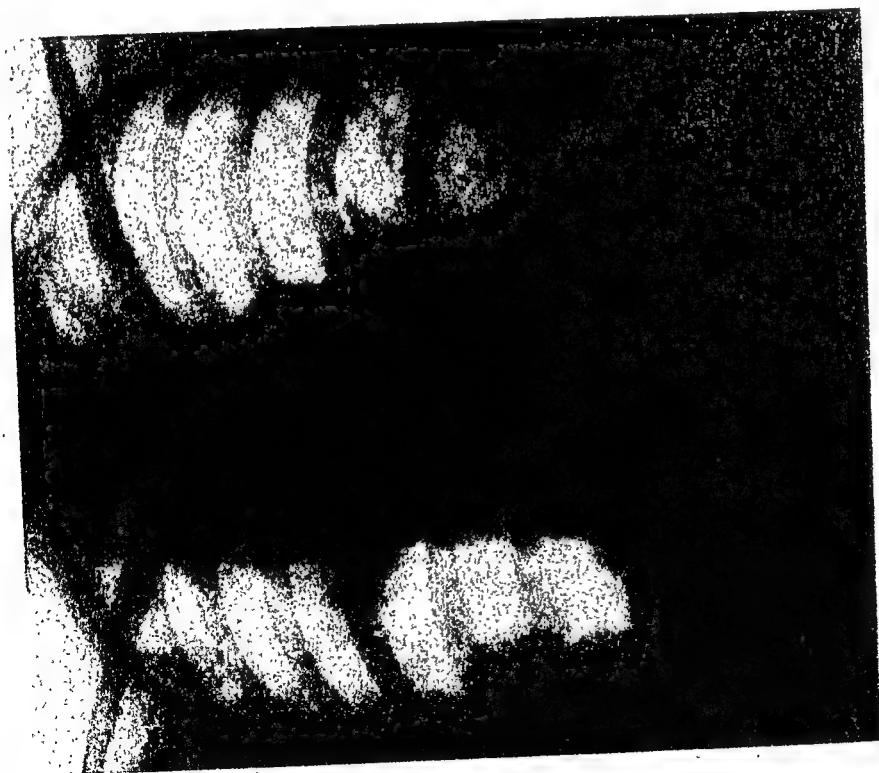


FIG. 305.—Endothelioma of pleura; diagnostic pneumothorax. Numerous nodular masses of growth project from the pleura.



FIG. 306.—Specimen to FIG. 305. Endothelioma of pleura.

pleura, presenting in the radiogram a clearly defined circular contour in the lower half of the left lung field. Autopsy revealed a thin-walled cyst, lined by a single layer of endothelium and containing clear fluid. A review of the literature is given in the article referred to.

CALCIFICATION OF THE PLEURA

Pleural calcification is usually easily recognised, in the form of irregular plaques or nodules immediately subjacent to the chest wall. It follows



FIG. 307.—Calcification of pleura, following old empyema.

injuries (hæmatoma) or infections of the pleural cavity. Occasionally it is extensive and forms an incomplete shell over the outer part of the lung, in which three lines can be recognised: (1) The anterior edge; (2) the posterior edge; and (3) the projection of the parietal surface. It is nearly always unilateral: clinical signs are those of thickened pleura.

The condition has been fully described by *Kjerp*, who collected 131 cases from the literature up to 1932.

CHAPTER XXXV

PNEUMOTHORAX, HYDROPNEUMOTHORAX, AND PYOPNEUMOTHORAX

PNEUMOTHORAX

THE ELASTIC recoil of the lung tends to separate the visceral pleura from the parietal pleura; this separation cannot actually take place, since the force is insufficient to create a vacuum. Contact is also said to be assisted to an extent difficult to determine by cohesion of the moist surfaces to one another. It is difficult to see how this force acts, since the surfaces glide over one another so freely; at all events, the introduction of the least quantity of air allows the surfaces to separate at once, and converts the potential space into an actual space.

The admission of air may be via the lung by rupture of the continuity of the visceral pleura (spontaneous pneumothorax), or through the chest wall from penetrating wounds, surgery, or the exploring needle (artificial pneumothorax).

Ætiology

Spontaneous Pneumothorax.—The rupture of the lung alveoli into the pleural cavity may result from various conditions: (1) Emphysema; (2) emphysematous bullæ; (3) tuberculosis with softening of the lung parenchyma; (4) in chronic fibrotic conditions, e.g. pneumoconiosis. The resulting pneumothorax may be limited to a portion of the cavity (e.g. apical localised pneumothorax in tuberculosis, limited by adhesions), or affect the whole cavity.

If the pleural aperture remains open, the condition is an open pneumothorax; usually it closes, and allows the air to be gradually reabsorbed. Occasionally it persists as a valvular opening, causing a great increase in the intrapleural pressure and dyspnoea. Such cases may clinically simulate acute abdominal disease. Spontaneous pneumothorax has been observed in newborn children (*Holz*). If bilateral, symptoms of asphyxia neonatorum may be present. Reabsorption is rapid and complete.

Artificial Pneumothorax.—Pneumothorax originating from an opening in the chest wall may also be open or closed. Examples of open pneumothorax are met with in penetrating wounds, and in drainage operations on the chest.

Clinical Features

The onset of a spontaneous pneumothorax is often sudden, and in a healthy athletic individual often occurs as a result of some sudden effort.

tensed muscles and closed glottis. In one personal case it occurred in an athletic young man while playing racquets, at the moment of "swiping" vigorously at a difficult shot. In another it occurred in a workman while straining at a large spanner, in tightening a nut in an awkward position. In a third patient it came on suddenly while he was blowing into a Woulfe's bottle, which had been prescribed as a breathing exercise. In all these cases the onset is timed by a sudden acute pain, followed immediately by collapse and marked dyspnoea.

In about one-half of the sixty-one cases observed by *Kerley* there was no history of violent effort, and the spontaneous pneumothorax occasionally occurred during rest.

Effects of Pneumothorax

The lung collapses in proportion to the amount of air admitted, by virtue of its elasticity, until its retractive force is balanced by the negative pressure in the pleural cavity. If there is a high intrapleural pressure, the lung is flattened against the heart, and may be quite invisible. As a rule the collapsed lobes are seen, not entirely airless, but partially emptied of air

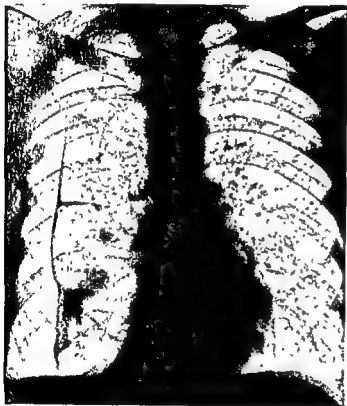


FIG 308 —Pneumothorax. Band like adhesions from lung to apex, and to diaphragm.

and blood. The vascular shadows diminish in size; while those of the opposite lung, now taking almost the entire pulmonary circulation, become broader and denser and show a richer network. The retracted lung enlarges with each inspiration. Outside the lung is a zone of absent lung-markings through which the detail of the ribs shows crisply. The ribs on the affected side run more horizontally, and the intercostal spaces are widened. The diaphragm is depressed and often shows reversed movement. The mediastinal structures are displaced



FIG. 309.—Artificial pneumothorax : fine adhesion at apex of upper lobe : cavities in both upper lobes.

Same case after division of adhesions : incomplete collapse of cavity.

towards the opposite side. Ballooning of the mediastinum is seen if the pressure is high. This may occur in two places: (a) *In the posterior mediastinum*; or (b) behind the upper part of the sternum—the more common site.

(a) *Danielius* has shown that at the retrocardiac weak spot the lungs are almost in contact with one another and only separated by the pleural leaves. In pneumothorax with high pressure a translucent area is seen here, passing across the midline, with a curved border. This is due to bulging of the posterior mediastinal pleura at the posterior weak spot, behind the œsophagus.

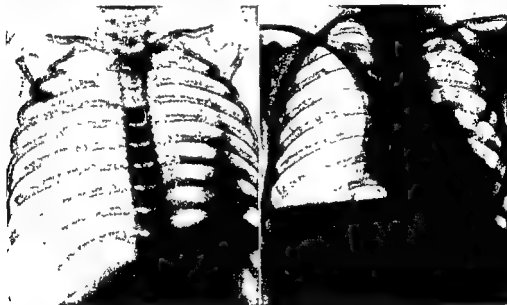


FIG 310—Spontaneous pyopneumothorax under pressure. Supine marked mediastinal hernia.

Same case, erect, mediastinal hernia still visible. Fluid level right base.

The trachea and œsophagus, in front of the weak spot, also deviate slightly from the midline.

(b) *Anteriorly* the anterior margin of the pleura may also cross the midline.

If fluid is present in the pneumothorax cavity, and the mediastinal "hernia" is large, a pocket fluid, showing a horizontal level, may occur on the *normal* side, in the lower part of the hernia.

In rare instances air may enter the space between the mediastinal and visceral pleura showing a translucent line bordering the heart (mediastinal pneumothorax).

Radiological Diagnosis

The diagnosis of pneumothorax is easy when uncomplicated by fluid; it is one of the few chest conditions which present quite typical appearances.

Difficulty arises only in partial pneumothorax, in incomplete pneumothorax, and in a few very rare conditions (extensive bullous emphysema, extensive congenital cystic disease). It is, however, quite easy to overlook a pneumothorax when the space is small—that is, when the lung has collapsed very little or has almost completely re-expanded. The lung in such cases may look almost normal, but careful inspection will show its edge as a faint line running internal to and parallel with the chest margin. It is extremely valuable to make the examination (especially fluoroscopy) in full inspiration and full expiration. In the latter phase the pneumothorax cavity becomes larger and more obvious.

By the same procedure the paradoxical movement of the diaphragm and of the mediastinum and heart may be observed. The direction of movement in both cases is towards the pneumothorax in inspiration. This does not occur in open pneumothorax. The movement of the mediastinum is then towards the sound side of inspiration, unless the opening in the chest wall is very small. If an opening is made into the pleural cavity through the chest wall in animals and a tube inserted, paradoxical movement of the mediastinum and diaphragm is observed when the tube is closed, normal movement when it is opened (*Assmann*).

Many complicated theories have been put forward to explain paradoxical movement of the mediastinum in pneumothorax, but it is easily explained if we remember that costal respiration enlarges both sides of the thorax, and that in the total space is suspended only one acting lung. This lung expands as a whole, filled with air at atmospheric pressure, and takes up as much of the available space as it can. It finds no rigid barrier in the midline, which it now transgresses; the mediastinum becomes bowed, and the mediastinal structures move towards the pneumothorax side. The air in the pneumothorax cavity is at the same time rarefied by the expansion of the thorax, and resists this encroachment with diminished force. During expiration the reverse occurs. We may regard the mediastinum as being sucked towards the pneumothorax in inspiration. Paradoxical movement of the diaphragm can be explained by a similar mechanism, assisted by the increased pressure in the abdomen which results from the downward movement of the normal diaphragm; but the reason for the apparent inertia of this strong muscular structure is unknown. It has been found by observation and by electrical records to be actually contracting at the moment of elevation.

LOCALISED PNEUMOTHORAX.—Localised pneumothorax may occur in the presence of adhesions either on the thoracic wall or at the apex, or even occasionally in an interlobar fissure. It may sometimes contain fluid (localised hydropneumothorax). The apical form is difficult to distinguish from apical cavity or a large apical bulla. Localised pneumothorax has been observed on the mediastinal surface of the lung.

Adhesions are occasionally seen in spontaneous pneumothorax in the form of fine strands leading out from a peak on the lung surface to some part of the

chest wall. They may be broad or delicate and are often multiple. They are best studied by stereoscopic radiograms.

BULLE.—On the surface of the collapsed lung in a case of pneumothorax, bullæ are sometimes observed (*Saul and Gordon*). These are sometimes circumscribed, about 3-7 cm. long, and have clearly defined contours both towards the pneumothorax and towards the lung itself. They lie on the outer surface of the upper lobe or apex.

Larger bullæ are sometimes seen with sharp external contours, ill-defined



FIG. 311.—Right apical pneumothorax.

towards the lung. These are subpleural collections of air resulting from interstitial emphysema.

THICKENING OF THE PLEURA.—This is occasionally visible as a broad zone of shadow either on the parietal or visceral pleura, most commonly in cases of air replacement of pleural effusions, or in cases of empyema after drainage.

FIBRIN BODIES.—These are rounded collections of fibrin, often seen in cases of air replacement of pleural effusions. They may be free, or attached, and sometimes calcify. One theory of their origin is that they result from small hæmorrhages during paracentesis.

Course

The course of spontaneous pneumothorax is favourable, and the air is completely reabsorbed in the average case in four to six weeks. In some cases within a fortnight. In exceptional cases the pneumothorax may persist for several years. Presumably in this case there is a pleuropulmonary fistula. The older the patient, the longer the time required for re-expansion. Although

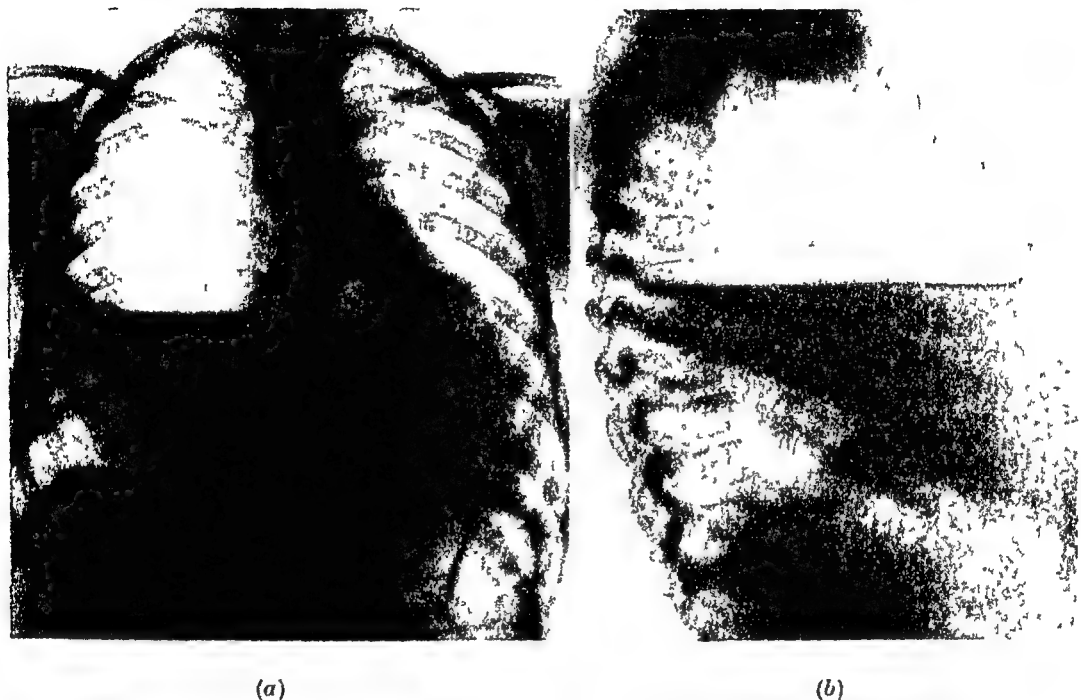


FIG 312.—(a) Pneumonia two weeks previously. Loculated pyopneumothorax under high pressure with mediastinal hernia. Fluid-level can be traced across midline. (b) Lateral view

a tuberculous aetiology is often suspected, very few of the patients ever develop tuberculosis.

HYDROPNEUMOTHORAX

Hydropneumothorax may arise : (a) By the secretion of pleural fluid into a spontaneous or artificial pneumothorax ; (b) by the admission of air by paracentesis, air replacement, or open operation into a thorax already containing pleural fluid ; (c) by rupture of an effusion, serous or purulent, into a bronchus ; or (d) by rupture of an abscess of the lung into the pleura.

The diagnosis is usually easy. The picture in films taken in the erect position, showing pneumothorax cavity, collapsed lung, and horizontal fluid-level, is quite typical. On screening, mediastinal displacement and paradoxical movements of mediastinum and diaphragm are recognised, and exaggerated pulsation of the unsupported heart and ripples on the surface of the

fluid may be seen. The film should be made with the tube centred as nearly as possible level with the top of the fluid. If this is not done the fluid-level may be ill-defined instead of sharp, or a double line may be seen, due to projection of the anterior and posterior edges of the fluid separately.

It is not possible to distinguish serous fluid from pus, unless the latter is very thick and moves sluggishly. The writer has never observed this sign. Thickening of the parietal pleura occurs more often and is more pronounced with empyema, but may occur with serous effusions.

A partial pneumothorax, with only a trace of fluid in the costo-phrenic angle, may be missed unless great care is used. Large hydro- or pyopneumothoraces may be overlooked, if, owing to the patient's condition, the examination is made in the supine position. The unilateral opacity due to the fluid, together with the displacement of the heart, may lead to a diagnosis of "pleural effusion," though physical signs may have pointed clearly to the presence of air as well as fluid. The mistake is obviated by the adoption of any manoeuvre capable of showing a fluid-level. Localised hydro- or pyopneumothorax may occur in any of the situations in which encysted effusions are found (parietal, diaphragmatic, mediastinal, or interlobar).

INTERLOBAR HYDROPNEUMOTHORAX.—Just as a communication between the lung and the main pleural cavity may lead to a pneumo- or hydropneumothorax, so may a penetration occur into the interlobar spaces, leading to the development of an interlobar pneumo- or hydropneumothorax. The presence of a horizontal fluid-level merely proves the existence of a cavity containing air and fluid, and only a most careful examination will determine whether this is intrapulmonary or interlobar. An interlobar hydropneumothorax may also arise from rupture of interlobar exudate into a bronchus, and in one personally observed case in which an interlobar effusion was topped by a small air bubble the cause was apparently infection with a gas-forming organism. Interlobar hydropneumothorax has to be distinguished from abscess, or other cavity.

The sharp demarcation of its contours, when carefully examined from all angles, is the chief point of distinction. If the case is one requiring surgery, the principal duty of the radiologist is the exact localisation of the lesion in relation to the chest wall, and the time available is best devoted to this end, especially if the patient is sick and has only limited powers of endurance. In this case an exact solution of the diagnostic problem may have to be subordinated to more practical considerations.

Hydropneumothorax after Air Replacement

In a series of cases of pleural effusion treated by air replacement by Williamson at the Manchester Royal Infirmary the writer had an opportunity of studying the radiological appearances. The lung was found to be collapsed to an extent which varied with the size of the effusion. This collapse was always more marked in the lower lobes. The shape of the collapsed lung was very



(b)

FIG. 313.—(a) Pneumococcal interlobar empyema. (b) Lateral view.

(a)

right side the differentiation is often a matter of great difficulty. Before rupture, while the shadow is still homogeneous, the upper domed contour looks exactly like that of an upwardly enlarged liver (tumour, cyst, hepatic abscess), or of the diaphragm pushed upwards by subphrenic abscess. In all these conditions diaphragmatic movements may be absent, though in intrahepatic conditions some movement is usually retained. After rupture when pyopneumo-



FIG. 315 — Post-pneumonic empyema. Pyopneumothorax. Thickening of parietal pleura, and, to a less extent, of visceral pleura.

thorax is present, the difficulty is to decide whether the dense line arching over it is the diaphragm itself, or the lower limit of the lung. In the latter case the upper border of the arched line is often blurred and ill-defined owing to condensation of the lung tissue by subpleural inflammation and pleural thickening; but this does not necessarily differentiate it from the diaphragm, for subphrenic infection is often accompanied by diaphragmatic pleurisy, which produces a similar shadowing above the diaphragm. Considerable help may be afforded by examination of the patient while he lies on the left side;

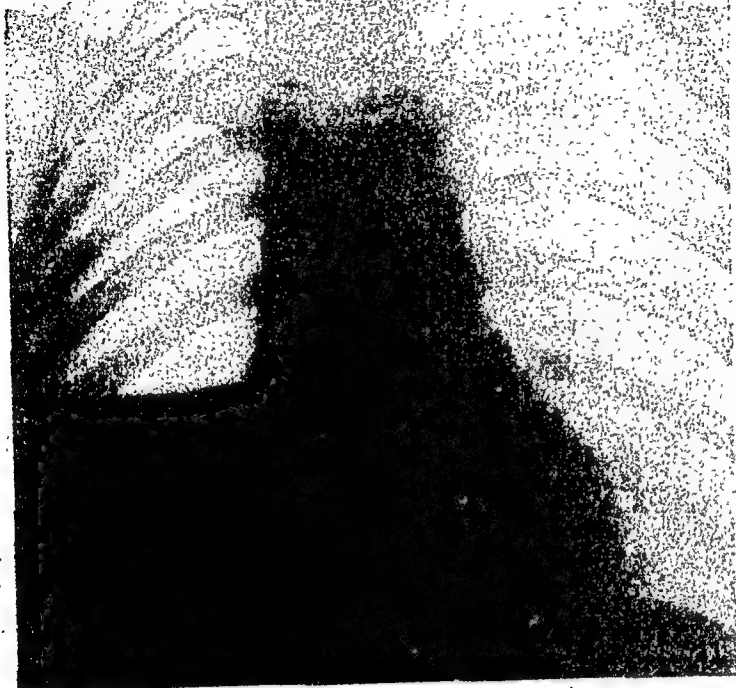


FIG. 316.—Hydropneumothorax—erect.



FIG. 316A.—Same case, lying on normal side. Note fluid level along the mediastinum, and thickened parietal pleura.

a postero-anterior or antero-posterior view in this position will show either (1) that the air bubble reaches down as far as the normal position of the diaphragm on the lateral chest wall and no farther (pyopneumothorax), see Fig. 317, or, (2) that it extends down into the abdomen (subphrenic abscess). This procedure may be supplemented by a lateral view with the patient supine; in this position an anterior subphrenic abscess containing gas shows a gas bubble extending downwards along the anterior abdominal wall in front of the liver.

These procedures have the advantage that they cause little disturbance of the patient. They are best carried out on a tilting couch, so that the head end may, if desired, be lowered. On the left side the problem is rendered a little simpler by the presence of the stomach, which, if it contains air, outlines the under surface of the diaphragm, and can be made to do so better by giving the patient an opaque or effervescent drink. If the diaphragm can be seen, the pyopneumothorax is easily localised correctly in the thorax. The lateral lying position is, however, even here, of distinct value, for if the patient lies with the left side uppermost and a postero-anterior view be taken, the air bubble of a subphrenic abscess, lying along the lateral abdominal wall, can be easily distinguished from the *magenblase*. From gas in the colon it is distinguished by the absence of haustra. The downward extent, and therefore the size, of the abscess can also be estimated by this method.

BIBLIOGRAPHY

- AARON, I., "Mediastinal Tumours and Malignant Lymphoma," *Radiology*, 1936, XXVI, 3, 605-621.
- ADAM, W. E., and VORWALD, A. J., "Treatment of Pulmonary Tuberculosis by Bronchial Occlusion," *Journ Thor Surg*, 1934, III, 6, 632-655.
- AEBY, "Der Bronchialbaum der Säugethiere und des Menschen," Leipzig, 1880.
- ALBRECHT, cit GHON, A., "Primary Lung Focus of Tuberculosis in Children," English edn, Churchill Ltd., 1916.
- ANDERSON, C. C., "X-ray Diagnosis of Hydatid Infection," Wellington Pub. Co., N.Z., 1926; "Difficulties and Fallacies in the Radiological Diagnosis of Hydatid Infection," *Journ Coll. Surg. Australia*, March, 1930.
- ANDERSON, E., "Über Anomalien der Wirbelsäule und Rippen," *Fortschr. Röntgenstr.*, 1926, XXIV, 4, 491.
- ANDREWS, J. R., "Planigraphy," *Amer. Journ. Roentgenol.*, 1936, XXXVI, 575.
- ANSON, B. J., and SMITH, H. V., "Accessory Pulmonary Lobe of Azygos Vein," *Amer. Journ. Roentgenol.*, 1936, XXXV, 631.
- ANSPACH, W. E., "'Military' Pulmonary Haemorrhages on Necropay Roentgenograms of Children," *Amer. Journ. Roentgenol.*, 1933, XXX, 6, 768; "Lower Lobe Collapse—Triangular Shadow at the Base of the Lung in Children," *Amer. Journ. Dis. Children*, May, 1934, XLVII, 1011; abstr *Amer. Year Book Rad.*, 1935, 111.
- ANSPACH, W. E., and WOLMAN, I. J., "Large Pulmonary Air Cysts in Infancy," *Surg. Gyn. and Obst.*, 1933, LVI, 635-645.
- ASCHOFF, *Verhandlung Deutsche Ges. f. Inn. Med.*, 1921, 13, cit. Fishberg, M., "Pulmonary Tuberculosis," 1932, I, 200.
- ASSMANN, H., "Frühinfiltrat," *Ergebnisse der gesamten Tuberkuloseforschung*, 1930, Bd. 1, 115-194; "Die klinische Röntgendiagnostik der inneren Erkrankungen," Berlin, 1934, 5th edn, 238; "Die Darstellung des Frühinfiltrats im Röntgenbild," *Fortschr. Röntgenstr.*, 1935, LI, 1.

- AUSTRIAN, C. R., "The Evolution of Tuberculous Infection in Childhood," *Amer. Rev Tuberc.*, 1927, XVI, 501-510.
- BARJON, F., "Radio-diagnosis of Pleuro-pulmonary Affections." Yale University Press, 1918, Humphrey Milford, London.
- BARNES, JN. M., and STEDEN, DAN. C., "Multiple Aneurisms of the Smaller Branches of the Pulmonary Artery." Buffalo, N.Y.
- BEAL, J. R., and GRAY, E. D., "A Case of Pulmonary Neoplasm of the Cavernous Type," *Brit. Journ. Rad.*, 1928, I, 151.
- BEAUMONT, G. E., "Massive Collapse of the Lung," *Brit. Med. Journ.*, Feb. 16, 1935, 314.
- BEAUMONT, G. E., and DODDS, E. C., "Recent Advances in Medicine," Churchill Ltd., 1936, 8th edn.
- BENTZEN, T. E., "A Case of Acute Siderosis," *Acta. Rad.*, 1933, XIV, 345.
- BERGENHOFF, W., "Interstitial Lung Syphilis," *Fortschr. Röntgenstr.*, 1930, XLII, 4, 478.
- BERTIN, E. J., "Pneumothorax in the Newborn," *Radiology*, 1936, XXVII, 584-593.
- BEUTEL, A., "Papillomatosis of Trachea and Main Bronchi," *Röntgenpraxis*, May, 1934, VI, 287.
- BLAINE, E. S., "Roentgenological Evidence of apparently healed Miliary Tuberculosis of the Lungs," *Amer. Journ. Roentgenol.*, 1924, XI, 233-237.
- BLAKE, F. G., and CECIL, R. L., "Studies on Experimental Pneumonia," *Journ. Exp. Med.*, 1920, XXXI, 403-445.
- BLAND-SUTTON, SIR JOHN, "Tumours," 7th edn., Cassell, 723.
- BLECHER, "Lungengangrän bei Bronchialstein," *Mitt. Grenzgeb. Med. Chir.*, 1928.
- BOCAGE, A. F. M., French Patent, 536464 (1922).
- BOLAND, C. R., and SHERET, J. E., "Post-operative Massive Collapse," *Lancet*, July 21, 1928, 111.
- BÖNNIGER, M., "Die Lungenspitze im Röntgenstereobild," *Fortschr. Röntgenstr.*, 1934, XIII, 638-677.
- BOWEN, D. R., "Acute Massive Collapse of the Lung," *Amer. Journ. Roentgenol.*, Feb., 1920, XXI, 101-141.
- BRADFORD, J. R., "Massive Collapse of the Lung," *Quart. Journ. Med.*, 1918-1919, XII, 127-150; "Massive Collapse of the Lung," Hunterian Lecture, *Lancet*, Feb. 15, 1930, 331.
- BRAILS福德, J. F., "Hydatid Disease in England," *Brit. Journ. Rad.*, Sept., 1931, IV, 45.
- BRINK and RIDDELL, cit. PANCOAST and PENDERGRASS.
- BRISCOE, J. C., "Mechanism of Post-operative Massive Collapse of the Lung," *Quart. Journ. Med.*, 1920, XIII, 203.
- BROMLEY, J., "Silicosis," *Brit. Journ. Rad.*, 1934, VII, 263.
- BROMLEY, J. F., "A Note on Hydatid Disease of the Chest," *Brit. Journ. Rad.*, 1933, VI, 237.
- BROWN, S., and MCCARTHY, J. E., "Intrathoracic Tumours," *Radiology*, 1936, XXVI, 574-585.
- BRUMPT, E., "Précis de Parasitologie," 1936.
- BRYCE, A. GRAHAM, and GRAY, E. D., "Unusual Difficulties in the Diagnosis of a Diaphragmatic Hernia," *Brit. Journ. Surg.*, 1933, XX, No. 80.
- BURRELL, L. S. T., "Recent Advances in Pulmonary Tuberculosis," Churchill Ltd., 1931.
- BURRELL, L. S. T., and MELVILLE, S., "Radiograms of the Chest (Clinical Interpretation of Aids to Diagnosis)," *Lancet*, 1930, II, 757-759, 813-814, 865-866.
- BURRELL, L. S. T., and TRAIL, R. R., *Lancet*, 1927, II, 1180.
- BURVILL-HOLMES, E., and BRODY, W., "Primary Angiofibroma of the Diaphragm," *Amer. Journ. Med. Sci.*, May, 1932, CLXXXIII, 679-680.
- CABOT, "A Case of Thymoma (Lymphoblastoma)," *New Eng. Med. Journ.*, Oct. 10, 1935, CCXIII, 726-728.
- CARTER, R. A., "Pulmonary Mycotic Infections," *Radiology*, May, 1936, XXVI, 5, 551-562.
- CARTY, J. R., and LIEBMAN, C., "The Roentgen Aspect of Empyema in Children," *Amer. Journ. Roentgenol.*, 1925, XIV, 215-218.

- CARVALHO, LOPO DE, "Die Angiopneumographie," *Beiträge zur Klinik der Tbk.*, 1932, Bd LXXX, Heft 5 und 6.
- CARVALHO, LOPO DE, and OTHERS, "Imagens Arteriais do Hilo e Topografia Arterial do Pulmão," *Jornal Lisboa Médica*, 1934, XI, 12, 923-940.
- CARVALHO, LOPO DE, and ROCHET, JOSÉ, "Topographie Arterielle du Poumon," *La Presse Médicale*, Dec, 1934, No. 102.
- CASTELLANI, A., "Fungi and Fungous Disease," 1928.
- CHAOU, H., *Sauerbruch. Chirurgie d. Brustorgane*, 1928, Bd I, 1; "Tomography, with Special Reference to Diagnosis of Pulmonary Conditions," *Fortschr. Röntgenstr.*, 1935, LI, 342; "Roentgenological Demonstration of Separate Layers of the Lung," *Beit. zur Klinik. Tbk. Brauer*, 1935, LXXXVI, 570.
- CHAOU, H., and GREINER, K., "Tomography of Carcinoma and Abscess of the Lung," *Fortschr. Röntgenstr.*, 1936, LIII, 232.
- CHERRY, H. H., "Bronchoectatic Emphysema of Adults," *Amer. Journ. Roentgenol.*, 1935, XXXV, 774.
- CHESTER, STEWART A., "The Evolution of Tuberculosis in the Human Lung," *Amer. Journ. Med. Sci.*, March, 1933, 346-350.
- CHRISTIE, A. C., "Bronchiectasis—Diagnosis and Treatment," *Radiology*, 1936, XXVI, 138-145; "Diagnosis and Treatment of Primary Cancer of the Lung," Sylvanus Thompson Lecture, *Brit. Journ. Rad.*, 1937, X, 141.
- CHRISTOPHERSON, J. B., "The Anatomy of Asthma as Disclosed by Lipiodol Investigation," *Lancet*, Jan. 7, 1933, 11-13.
- CLAESSENS, G., "The Roentgen diagnosis of Echinococcus Tumours," *Acta. Rad.*, Suppl., 1928, VI, Stockholm; "On Echinococcus of the Lung," *Acta. Rad.*, 1935, XVI, 601.
- CLARKE, B. E., "Superior Pulmonary Sulcus Tumour (Pancoast)," *Amer. Journ. Path.*, 1934, X, 603.
- COMROE, B. I., "Hemopericardium: Report of Case of Unknown Etiology with Recovery following 2 Pericardial Paracenteses," *Amer. Heart Journ.*, June, 1934, IX, 680-685.
- CONNOLLY, A. E., "Superior Pulmonary Sulcus Tumour," *Brit. Journ. Rad.*, Dec., 1935, VIII, No. 96, 781.
- COOKE, W. E., "Pulmonary Asbestosis," *Brit. Med. Journal*, 1927, II, 1024.
- CORTYLOS, P. N., and BIRNBAUM, G. L., "Lobar Pneumonia, Pneumococcal Lobar Atelectasis," *Arch. Surg.*, 1929, XVIII, 190; *Amer. Journ. Roentgenol.*, 1929, XXII, 401.
- COLTARD, H., and BACLES, F., "Roentgen diagnosis of Epithelioma of Larynx and Pharynx," *Amer. Journ. Roentgenol.*, Sept., 1932.
- CROSWELL, C. V., and KING, J. C., "Congenital Air Cyst of the Lung," *Journ. Amer. Med. Assn.*, 1933, CI, 832-834.
- CUMMINS, S. L., "Pneumoconiosis with Special Reference to Silicoantracosis of Coal Miners," *Brit. Med. Journ.*, Aug., 1935, II, 287-290.
- CUNNINGHAM, D. J., "Textbook of Anatomy," Edited by J. C. Brash and E. B. Jamieson, 1937, 7th edn., London, H. Miford (Oxford Med. Publications).
- DAAN, A., "Der Lobus Venæ azygos im Röntgenbilde," *Acta. Rad.*, 1933, XIV, 4, 375.
- DANIELIUS, G., Zur Frage des "Pleuritischen Mediastinalstreifens," *Fortschr. Röntgenstr.*, 1933, XLVII, 271.
- DAVIDSON, M., "Cancer of the Lung," London, 1930.
- DAVIES, D., HODGSON, H., GRAHAM, and WHITBY, L. E. H., "Study of Pneumococcal Pneumonia," *Lancet*, Apr., 1935, I, 791-796, 849-853, 919-924.
- DAVIES, H. M., "Importance of Radiology in Diagnosis of Pulmonary Tuberculosis," *Brit. Med. Journ.*, Aug. 27, 1927, 335.
- DAVIES, K. S., "Roentgen Changes following the Introduction of Mineral Oil into the Lung," *Radiology*, Feb., 1936, XXVI, 13-136.
- DEBRÉ, R., and GILBRIN, E., "Sur les Kystes gazeux du poumon et les bronchiectasies," *Presse méd.*, July 11, 1934, XLII, 1113-1116.
- DELHERM, L., and CHAPERON, R., "Les Ombres Hilaires," *Bull. Soc. Radiologie*, Nov 1922, "Comment doit on interpréter les ombres hilaires normales?" *J. de radiol. et d'électrol.*, Paris, 1923, VII, 329-334.

- SKER, H., "Die Röntgendiagnostik in der Tuberkulose-fürsorgestelle," *Klin. Wochenschrift*, Jan. 30, 1932, XI, 207-209.
- ESJARDINS, A. U., "Reaction of Pleura and Lungs to Roentgen Rays," *Amer. Journ. Roentgenol.*, Nov. 1926, XVI, 444-448.
- ESJARDINS, A. U., HABEN, H. C., and WATKINS, C. H., "Unusual Complications of Lymphoblastoma," *Amer. Journ. Roentgenol.*, Aug., 1936, XXXVI, 2, 169.
- ETIMERS, H., "Zur Kasuistik Kongen. Kysten. in. d. Lunge," *Acta. Rad.*, 1931, XII, 135-139.
- EVÉ, F., "L'Echinococcose Secondaire," Thèse de Paris, 1902; "Echinococcose Secondaire du Poumon," *Comptes Rendues, Société de Biologie*, 16 Juillet, 1904, and Tome CVII, 438; *Arch. méd.-chir. de l'app. respir.*, 125-140; "Kystes Hydatique du Poumon," *Arch. Med. de l'Appareil Resp.*, 1926, Tome I, No. 2.
- EW, H., "Hydatid Disease," Sydney, 1928; "Advances in our Knowledge of Hydatid Disease," *Brit. Med. Journ.*, Oct. 5, 1935, 620.
- FIENST, C., "Zur Lungensyphilis der Erwachsenen," *Röntgenpraxis*, Aug., 1932, Heft 16, IV, 703-710.
- FIETLEN, H., "Über Interlob. Pleuritis," *Erg. d. Inn. Med.*, 1913, XII, 196.
- FOIG, A. T., "Dysphagia due to Unilateral Pulmonary Fibrosis," *Brit. Med. Journ.*, March 7, 1936.
- FOIG, A. T., and McLAUGHLIN, A. I. G., "X-ray Appearances of the Lungs in Electric Arc Welders," *Lancet*, Apr. 4, 1936, 771-775.
- DONALDSON, S. W., "Hyperplasia of the Thymus—Clinical and Radiological Study of 1,045 patients, including the family history," *Amer. Journ. Roentgenol.*, XXIV.
- DOUB, H. P., "Roentgendiagnosis and Treatment of Thymomata," *Radiology*, 1930, XIV, 267-281.
- DOUGLAS, B. H., "Roentgen Examination in Pulmonary Tuberculosis," *Amer. Journ. Roentgenol.*, 1933, XXX, 306.
- DOWLING, G. B., "Blastomycosis," *Brit. Ency. Med. Practice*, Rolleston, II, 403. Butterworth, London, 1936.
- DOWN, E. E., "Lung Changes subsequent to Irradiation in Cancer of the Breast," *Amer. Journ. Roentgenol.*, 1936, XXXVI, 61-64.
- DUBROW, J. L., *Radiology*, April, 1925, XXIV, 480.
- DUGUID, J. B., "The Incidence of Intrathoracic Tumours in Manchester," *Lancet*, 1927, II, 111-116.
- DUNHAM, H. K., "X-ray Examination of the Chest and an X-ray Classification of Pulmonary Tuberculosis," *Amer. Journ. Roentgenol.*, 1921, VIII, 427.
- DUNHAM, H. K., and SKAVLEM, J. H., "A Comparative Study of the Pathology and X-ray Densities of Tuberculosis Lung Lesions," *Amer. Rev. Tuberc.*, 1921, V, 278-322; "Sinus Disease and Lung Infections," *Journ. Radiology*, 1923; "Applied Pathology of Pulmonary Tuberculosis," *Radiology*, March, 1925.
- DWYER, M. F., "Report of a Case of Thymoma," *Radiology*, Dec. 1927, IX, 510.
- EDWARDS, A. TUDOR, "Intrathoracic New Growths," *Brit. Journ. Surg.*, April, 1927, XIV, 56, 607; "Surgical Treatment of Intrathoracic New Growths," *Brit. Med. Journ.*, 1932, I, 827-830.
- EDWARDS, A. TUDOR, and OTHERS, "Discussion on Intrathoracic Tumours," *Proc. Roy. Soc. Med.*, 1929-1930, XXIII, 689-701.
- EISLER, "Interlobäre-pleuritische. Schwarte," *Münch. Med. Wochschr.*, 1912, 1899.
- ELIASBERG, H., and NEULAND, W., "Die Epituberkulöse Infiltration der Lunge Säuglingen und Kindern," *Jahrb. f. Kinderh. Berlin*, 1920, XCIII, 88-97; "Zur Klinik der epituberkulösen und gelatinösen Infiltration des kindlichen Lunge," *Jahrb. f. Kinderh. Berlin*, 1921, XCIV, 102-118.
- ELLIOTT, T. R., and DINGLEY, L. A., "Massive Collapse of the Lung following Abdominal Operations," *Lancet*, 1914, I, 1305-1309.
- ELLIS, R. W. B., "Atelectatic Bronchiectasis in Childhood," *Arch. Dis. Childhood*, 1931, VIII, 25.
- ELLMAN, P., "Pulmonary Asbestosis," *Brit. Journ. Rad.*, May, 1934, VII, 77, 281.

- ELOESSER, L., "Congenital Cystic Disease of the Lung," *Surg. Gyn and Obsl.*, 1931, LII, 747-758; *Radiology*, 1931, XVII, 912-929.
- ELWARD, J. E., "Congenital Aplasia of Lung," *Radiology*, 1936, XXVII, 6, 667-671.
- ENGEL, ST., and SCHALL, L., "Handbuch der Röntgen-diagnostik und Therapie im Kindesalter," Geo. Thieme, Leipzig, 1933
- ENGEL, ST., and SAMSON, *Ibid.*
- EXTIN, M., "Ueber Pneumothorax," *Fortschr Röntgenstr.*, 1915, XXIII, 10-30.
- ERLICH, D. F., and HAUPTMAN, H. A., "Primary Carcinoma of the Lungs," *Radiology*, 1936, XXVI, 3, 563.
- ETTING, F., "Pleuritis mediastinalis und Infiltration eines Lungenlappens (lobus infracardiacus)," *Monatschr f. Kinderh., Leipz.*, 1924, XXVIII, 207-214
- EVANS, W. A., and LEUCUTIA, T., "Intrathoracic Changes Induced by Heavy Radiation," *Amer Journ Roentgenol*, March 1925, XIII, 203-220
- EWART, W., "The Bronchi and Pulmonary Blood Vessels," 1889, J & A Churchill, Ltd, London
- EWING, J., "Neoplastic Diseases," 2nd edn, 1922, Saunders, Philadelphia
- EVERT, J. A., *cit DOUB.*
- GARISSAS, P. L., "Serial Bronchographs in Early Diagnosis of Bronchial Carcinoma," *Fortschr Röntgenstr.*, 1933, XLVIII, 330
- FAULKNER, W. B., JR., and FAULKNER, E. C., "Diagnostic Pneumothorax. Aid in Diagnosis of Pleural Tumours," *Radiology*, 1932, XVIII, 1023-1027.
- FAWCETT, R., "Fungoid Conditions of the Lungs," Pt II, *Brit Journ. Rad.*, 1936, IX, 354.
- FENZL, N. S., "Late X ray and Radium Effects," Incidence, Etiology and Medical Treatment," *Brit Journ Rad.*, 1933, VI, 148-157
- FIHERO, MAURICE, "Pulmonary Tuberculosis," 1932, 4th edn., Henry Kimpton, London.
- FLISCHNER, F., "Das Röntgenbild d interlob. Pleuritis," *Urg d Mediz Strahlforsch., Leipzig*, 1926, Bd II, 199-248. ' Lungenapiznenbefunde im Röntgenbild,' *Fortschr. Röntgenstr.*, 1927, XXXV, 749-755. ' Der sichtbare Bronchialbaum, ein differential-diagnostisches Symptom im Röntgenbild der Pneumonie,' *ibid.*, 1927, XXXVI, 310-323; ' Über das Wesen der basalen horizontalen Schattenstreifen im Lungenfeld,' *Wien Arch f inn Med.*, 1936, XXVIII, 461; ' Atelektase und gerichteter Kollaps der Lunge,' *Fortschr Röntgenstr.*, 1936, LIII, 607
- FOCKE, F., "Ueber die cartilaginösen Pleuraschwien an der Lungenspitze," *Beit z. Klin d Tuberk., Berlin*, 1924, LIX, 228-239
- FREEDMAN, E., "Congenital Cysts of the Lungs," *Amer Journ Roentgenol*, Jan., 1936, XXXV, No 1, 44-52
- FREEDMAN, E., and HIGLEY, C. S., "Syphilitic Gamma of the Lung," *Amer Journ Roentgenol*, 1934, XXXI, 333
- FREEDMAN, E., and SIMON, M., "Simple Cyst of the Pleura," *Amer Journ Roentgenol*, 1936, XXXV, 53; "Emphysematous Blebs and Bullae," *ibid.*, 324-333.
- FREUND, F., "Ueber den Einfluss der Röntgen-Frühdiagnose auf die Tuberkulose-Mortalität," *Wien klin Wchnsch.*, 1936, XLIX, 1096-1098
- FRIED, B. M., "Primary Carcinoma of the Lung," Williams & Wilkins Co., Baltimore, 1932. "Bronchogenic Cancer, Combined with Tuberculosis of the Lungs," *Amer. Journ Cancer*, 1935, XXIII, 247-266
- FRUCHTER, J. M., "Discrete Pulmonary Lesions Roentgenologically Considered," *Radiology*, Nov., 1935, XXV, 609
- FULLER, C. B., and JAGGER, F. I. C., "Case of Dermoid Cyst at Back of Chest," *Brit. Journ Rad.*, May, 1927, XXXII, 176
- GARCIN, J., "Anatomie Radiol des Pouxons," *Journ d Radiologie et Electologie*, 1924, VIII, 8.
- GARLAND, L. H., "X-ray Aspects of Pneumococcosis," *Radiology*, 1936, XXVII, 21-32.
- GASKELL, J. F., "Experimental Pneumococcal Infections of the Lung," *Journ. Path. and Bact.*, 1925, XXVIII, 427; "Bradshaw Lecture on the Pathology of Pneumonia," *Lancet*, 1927, II, 951.

- GHON, ANTON, "Primary Lung Focus of Tuberculosis in Children," English edn., Churchill Ltd., 1916.
- GIBSON, D. N., "Congenital Cystic Lung—case report," *Amer. Journ. Roentgenol.*, 1929, XXII, 155-157.
- GLASS, A., "Broncho-pulmonary Segments with Special Reference to Lung Abscess," *Amer. Journ. Roentgenol.*, March, 1934, XXXI, 328.
- GOLD, E., "Case of Thymus Tumour with Myasthenia Gravis," *Wien. Med. Wochschr.*, May, 1935, XLVIII, 694-698.
- GOLDEN, R., "The Effect of Bronchostenosis upon the Roentgen-ray Shadow in Carcinoma of the Bronchus," *Amer. Journ. Roentgenol.*, 1925, XIII, 21; "Diagnostic Roentgenology," Thos. Nelson & Sons, New York and Edinburgh.
- GORLITZER, V., "Neurofibromatosis Recklinghausen excessiva und Skelettmissbildung," *Arch. f. Dermat. u. Syph.*, 1930, CLIX, 510-522.
- GRABERGER, G., "Beitrag zur Kenntnis der basalen paramediastinalen Dreieckschatten," *Acta. Rad.*, 1931, XII, 240-253.
- GRAEF, I., and STEINBERG, I., "Superior Pulmonary Sulcus Tumour," *Amer. Journ. Roentgenol.*, 1936, XXXVI, 293.
- GRAEFF and KÜPFERLE, "Die Lungenphthise," Berlin, 1923.
- GRANDY, C. C., "Roentgenographic Demonstration of Adenoids," *Amer. Journ. Roentgenol.*, 1925, XIV, 114.
- GRAWITZ, P., "Über angeborene Bronchiektasie," *Virchow's Arch. für Path. Anat.*, 1880, LXXXII, 217.
- GREINER, K., "Tomography of the Normal Lung," *Fortschr. Roentgenstr.*, 1935, III, 443.
- GRETHMANN, W., "Architecture of Terminal Bronchi of Human Lung," *Amer. Rev. Tuberc.*, 1935, XXXI, 3, 261-298.
- GROOVER, T. A., "Intrathoracic Changes following Roentgen Treatment of Breast Carcinoma," *Amer. Journ. Roentgenol.*, 1923, X, 471.
- GROSSMANN, G., "Lung Tomography," *Brit. Journ. Rad.*, 1935, VIII, 733.
- GROTH, K. E., "Roentgen picture of Epipharynx of Children," *Acta. Rad.*, 1934, XIV, 5, 464.
- GRUBER, cit. STOLOFF.
- GULEKE, M., "Über Sanduhrgeschwülsten der Wirbelsäule," *Arch. Klin. Chir.*, 1922, CXIX, 833; 1930, CLXI, 710; cit. Schmorl. "Ges. und kranke Wirbelsäule," G. Thieme, Leipzig, 1932.
- HADFIELD, G., and GARROD, L. P., "Recent Advances in Pathology," J. & A. Churchill. Ltd., 2nd edn., 1934.
- HAMMARSKJÖLD, B., "Contribution to Knowledge of Teratoma and Dermoids in the Anterior Mediastinum," *Acta. Rad.*, 1934, XV, 210-224.
- HAMPTON, A. O., and KING, S., "The Middle Lobe of the Right Lung," 1936. *Amer. Journ. Roentgenol.*, XXXV, 721-739.
- HARRINGTON, S. W., "Surgical Treatment of Tumours of Lung and Mediastinum," *Surg. Gyn. and Obst.*, 1931, LII, 417-425; "Surgical Treatment in 11 Cases of Mediastinal and Intrathoracic Teratomas," *Journ. Thor. Surg.*, 1933, III, 50-72; "Surgical Treatment in 14 Cases of Mediastinal or Intrathoracic Perineural Fibroblastoma," *Journ. Thor. Surg.*, 1934, III, 6, 590-611.
- HARRISON, R. STEWART, "Mediastinal Tumours and Leucaemia," *Acta. Rad.*, 1934, XV, 539.
- HART, F. D., and ELLISON, P. O., "Mediastinal Ganglioneuroblastoma," *Lancet*, June 19, 1937, 1438.
- HARTFALL, S. J., and PYRAH, L. N., "Mediastinal and Apical Empyema," *Brit. Med. Journ.*, Dec., 1934, 1039.
- HARTUNG, A., and FREEDMAN, J., "Pulmonary Syphilis—Three Cases in Adults with Particular Reference to Roentgen Aspects," *Journ. Amer. Med. Ass.*, 1932, XCVIII, 1969-1972.
- HARTUNG, A., and RUBERT, S. R., "Roentgen Aspects of Sympathetic Neuroblastoma," with report of 2 cases, *Radiology*, May, 1935, XXIV, 607.

- HAUDEK, M., "Erg. fortlaufend Roentgenbeobachtung. bei Lungen Tbk.," *Acta. Rad.*, 1926, VI, 502; "Über die Bedeutungslösigkeit der Spitzen Tbk.," *Fortschr. Röntgenstr.*, 1927, XXXVI, and *Acta. Rad.*, 1926, VI, 502; "Über die verhältnismässig Bedeutungslösigkeit der Spitzentuberkulose," *Fortschr. Röntgenstr.*, 1927, XXVI, Verh. dtsch. Röntgen.—Ges. 18 Kongress, 1927; "Durchwanderungspleuritis bei abdominalen Krankheitsprozessen," *Fortschr. Röntgenstr.*, 1932, XLV, 1-8.
- HÉBERT, PIERRE, "L'Echinococcose secondaire de P. Dévé en pathologie humaine," Paris, 1932
- HEDBLUM, C. A., "Intrathoracic Dermoid Cysts and Teratomata," *Journ Thor. Surg.*, 1933, III, 22-49.
- HEFFERNAN, P., and GREEN, A. T., "The Method of Action of Silica Dust in the Lungs," *Journ of Industrial Hygiene*, 1928, X, 272-278
- HOLER, C., and HOLTHUSEN, H., "Hæmatogenous Disseminate Pulmonary Tuberculosis with Complete Cure," *Röntgenpraxis*, March, 1936, VIII, 161-168.
- HEUER, G. J., "Exlubation of Patient with a Calcified Cyst in the Right Pleural Cavity," *Bull Johns Hopkins Hosp.*, 1917, n.s., XXVIII, 153; "Thoracic Tumours," *Arch. Surg.*, 1929, XVIII, 271.
- HEUER, ROBERTSON, and BROWN, cit. MÖR.
- HICKEY, P. M., and SIMPSON, W. M., "Primary Chondroma of the Lung," *Acta radiol.*, Stockholm, 1926, V, 475-500.
- HIRSCH, I. SETH, "Pulmonary Changes in Polycythemia," *Radiology*, 1936, XXVI, 460.
- HOCHSINGER, see ASSMANN, "Röntgendiagnostik Inn. Erkrankungen," 1934, I, 194.
- HODGSON, GRAHAM, "Large Metastasis in Chest simulating a Dermoid," *Brit. Journ. Rad.*, 1933, VI, 478
- HOLMES, A. H., "Pulmonary Carcinoma from the Clinical Standpoint," *Med Press and Circular*, 1936, CXCII
- HOLZ, I., "Beitrag zur Frage des Spontanpneumothorax bei Neugeborenen," *Kinderärztl. Praxis*, Jan., 1936, VII, 3-9.
- HOTZ, "Zur Kenntnis d. Interlob. Schwarten im Roentgenbild," *Fortschr. Röntgenstr.*, 1929, XXVII, 384.
- HOYLE, C., and VAIZEY, M., "Chronic Miliary Tuberculosis," Maford: Oxford University Press, 1937
- HULTEN, "Ellenbeinwiabel bei Lymphogranulomatose," *Acta Rad.*, 1927, 7, 243
- HUSTEN, K., "Über den Lungenasinus," *Beitr. z. Path. Anat.*, Bd. 68.
- IKEDA, KANO, "Bronchopulmonary Moniliasis and its Relation to Obscure Pulmonary Infection," *Archives of Pathology*, July, 1936, 62-81.
- ILES, A. J. G., "Case of Thoracic Fibroma," *Brit. Journ. Rad.*, May, 1932, V, 63, 460
- ILLINGWORTH, and DICK, "Textbook of Surgical Pathology," J. & A. Churchill, Ltd, 1931
- JACCHIA, P., "Phlebeetasia in Parenchyma of the Lungs," *Acta. Rad.*, 1936, XVII, 74-78.
- JACKSON, C., "Endoscopy," Gaston Dine, Paris, 1924.
- JACOBUS, H. C., "Case of Severe Bullous Emphysema," *Acta. Rad.*, 1933, XVI, 661-667.
- JACOBUS, H. C., and KEY, E., "Some Experiences of Intrathoracic Tumours, their Diagnosis and their Operative Treatment," *Acta Chir. Scand.*, Stockholm, 1921, LIII, 573-619.
- JACOBUS, H. C., and WESTERMARK, N., "Further Study of Massive Collapse of Lung," *Acta. Rad.*, XI, 547; "Contribution to the Interpretation of Localised Roentgen Opacities in the Neighbourhood of the Interlobar Fissures of the Lungs," *Acta. Chir. Scand.*, 1932, LXXI, 494-516.
- JACOX, H. W., "Superior Pulmonary Sulcus Tumour," *Journ Amer. Med. Assn.*, 1934, CIII, 84-87.
- JACQUEROD, "Étude Clinique et Radiologie des Cavernes Tuberculeuses," Masson et Cie, Paris, 1938.
- JACQUEROD and ELLMAN, "Radiological Appearances of Tuberculous Pneumonia and Tuberculous Bronchopneumonia," *Brit. Journ. Rad.*, Oct., 1927, XXXII, 327.
- JÄDERHOLM, K. B., "Ein Fall von Bullösem Emphysema," *Acta. Rad.*, 1932, XIII, 51; "Über perikard divertikel," *Acta. Chir. Scand.*, 1932, LXXI, 517-523.

- JÄNSSON, G., "Pericardial Diverticulum," *Acta. Rad.*, 1931, XII, 50-57; "Method for Roentgen-examination of Hypopharynx and Upper Air Passage," *ibid.*, 1934, XV, 125; "Die Röntgendiagnose bei Intrathorakalen Neurinom," *ibid.*, 411; "Some Roentgenological Observations regarding Pulmonary Silicosis in Porcelain Workers," *ibid.*, 1935, XVI, 431.
- JOHNSTON, see MANGES.
- JONES, C., "Rib Defects simulating Pulmonary Cavitation," *Radiology*, Nov., 1935, XXV, 5, 533.
- JONES, W. R., "Silicotic Lungs, the Minerals they Contain," *Journ. Hygiene*, 1933, XXXIII, 307-329.
- KAESTLE, K., "Über die Pneumonokoniose der Sandstein-, Kieselgreise-, Granit-, Muschelkalk-, und Zementarbeiter," *Fortschr. Röntgenstr.*, 1928, XXXVIII, 1016; "Über Pneumonokoniosen," *Fortschr. Röntgenstr.*, 1932, XLVI, 117-125.
- KAHLER, "Über Divertikel des Tracheo-Bronchialbaumes," *Mtschr. Ohrenheilk.*, 1911, 45, cit. Schinz.
- KARSNER, HOWARD T., "Human Pathology," Lippincott & Co., Philadelphia and London, 1935.
- KAESTLE in SCHITTENHELM, A., "Lehrbuch der Röntgendiagnostik," 1924, 395.
- KATS, E. G., "Malignant Thymoma with Metastasis into Pericardium," *Trach. delo.*, 1934, XVII, 683-684.
- KAUNITZ, J., "The Three Zones of Simple Pleural Effusions," *Amer. Journ. Roentgenol.*, 1936, XXXV, 1, 57-58.
- KAUTZ, F. G., and PINNER, M., "Extrapericardial Fat Bodies," *Amer. Journ. Roentgenol.*, Jan., 1936, XXXV, 1, 40.
- KEIBEL, F., and MALL, F. P., "Manual of Human Embryology," J. B. Lippincott & Co., Philadelphia and London, 1912, II, 485 and 492.
- KELLER, W. L., and CALLENDER, G. R., "Neurofibroma Arising on Pericardial Pleura," *Ann. Surg.*, 1930, XCII, 666-672.
- KERLEY, P., "Neoplasms of the Lungs and Bronchi," *Brit. Journ. Rad.*, 1925, XXXVI, 333; "Fibrocystic Disease of the Lung," *Brit. Journ. Rad.*, 1927, XXXII, 245-249; "The Inferior Accessory Lobe of the Lung," *Brit. Journ. Rad.*, 1932, V, 234-240; "Emphysema," *Proc. Roy. Soc. Med.*, Aug., 1936, XXIX, 53-61; "Congenital Atelectasis in a New-born Child," *Brit. Journ. Rad.*, 1936, IX, 481; "Recent Advances in Radiology," J. & A. Churchill, Ltd., 2nd edn., London, 1936.
- KERLEY, P. J., SHORE, L. R., and YOUNG, W. A., "A Case of Fibrocystic Disease of the Lung," *Lancet*, Oct. 1, 1927, II, 699-701.
- KETTLE, E. H., "Experimental Silicosis," *Journ. Ind. Hygiene*, 1926, VIII, 491-495.
- KIENBÖCK, R., and MAYER, S. A., "Über Multipel Neurofibromatose," *Fortschr. Röntgenstr.*, 1935, LI, 4, 387.
- KIENBÖCK and WEISS, "Diverticulum of Pericardium," *Fortschr. Röntgenstr.*, Nov., 1934, L, 442.
- KIRKLIN, B. R., "Congenital Cysts of the Lung from the Roentgenological Viewpoint," *Amer. Journ. Roentgenol.*, July, 1936, XXXVI, 1, 19-29.
- KIRKLIN, B. R., and PATERSON, J. RALSTON, "Roentgenologic Manifestations of Primary Carcinoma of the Lung," *Amer. Journ. Roentgenol.*, 1928, XIX, 20 and 126.
- KJAERGAARD, "Spontaneous Pneumothorax in the Apparently Healthy," *Acta. Med. Scand.*, (Suppl.) 1932, XLIII, 35.
- KJERF, G., "Pleural Calcification," *Acta. Rad.*, 1932, XIII, 334-348.
- KLEHMET, M., "Zur Diagnose der Pneumonokoniosen," *Beit. z. Klin. d. Tuberk.*, Berl., 1920, XLVI, 153-173.
- KLEMPERER, P., and RABIN, C. B., "Primary Neoplasms of Pleura, Report of 5 Cases," *Arch. Path.*, 1931, XI, 385-412.
- KNUSSON, F., "On Intrathoracic Neurinomata," *Acta. Rad.*, 1931, XII, Fasc. 4, 388; "Zur Kenntnis der Pleura Parietalis," *Acta. Rad.*, 1932, XIII, 638-677.
- KÖHLER, ALBAN, "Röntgenology," 5th edn., Trans. Arthur Turnbull, Baillière, Tindall & Cox, London, 1929.

- KONSTAM, G. L. S., and TURNBULL, H. M., "Syphilia of the Lung with Ayerza's Disease," *Lancet*, Oct. 12, 1929, 756.
- KOONTZ, A. R., "Congenital Cysts of the Lungs," *Bull. Johns Hopkins Hosp.*, 1925, XXXVII, 340-361.
- KOPSTEIN G., "Zur Röntgenologischen Symptomatologie von Mittellappenerkrankungen," *Fortschr. Röntgenstr.*, 1933, XLVIII, H. 2, 145.
- KORNBLUM, K., "Roentgendiagnosis of Pulmonary Infection with Friedländer Bacillus," *Amer. Journ. Roentgenol.*, 1928, XIX, 513.
- KORNBLUM, K., and OSMOND, L. H., "Mediastinitis," *Amer. Journ. Roentgenol.*, 1934, XXXII, 23-42.
- KRAUSE, A. K., "Experimental Studies on Tuberculous Infection," *Amer. Rev. Tuberc.*, 1922, VI, 1-20.
- KRAUSE, cit. ASMANN.
- KRAUSE, K., and LOBEN, F., "Pneumokoniose und ihre Abgrenzung gegen Tuberkulose," *Deut. z. Klin. d. Tuberk.*, 1927, LXVII, 369-385.
- KREUZGUTH, S., and SCHUMACHER, O., "Die Topographischen Verhältnisse der Interlobären Spalten der Lunge," *Acta Rad.*, 1921, I, 234.
- KUHLMANN, F., "Honeycomb Lung," *Fortschr. Röntgenstr.*, Oct., 1933, LII, 402-407.
- KUSN, cit. GHON.
- LAMBERT, S. W., and KNOX, L. C., "Intrathoracic Teratomata," *Trans. Assn. Amer. Physicians*, 1920.
- LANGE, C. DE., "Congenital Cystic Lung and Agenesis of Bronchiectasia," *Nederl. Tijdschr. v. Geneesk.*, June 12, 1926, I, 2515-2524, "Angeborene Zystenlunge und agenetische Bronchiektasie," *Acta Paediatrica*, 1927, VI, 352-372.
- LANGE, C. DE., and BRUIN, M. DE., "Diagnosis of Congenital Cysts of Lung," *Nederl. Tijdschr. v. Geneesk.*, Nov. 10, 1934, LXXVIII, 5127-5136.
- LAURELL, H., "Deutung der Ringschatten in den Lungen," *Acta Rad.*, 1929, X, 72-119; "Der Nachweis minimaler Pleuraexsudate; Differentialdiagnose bei lamellärer Pleuritis," *Acta Rad.*, 1935, XVI, 691.
- LAURELL and HULTEN, see FLEISCHNER, F., "Plattenförmige Atelektasen in den Unterlappen d. Lunge," *Fortschr. Röntgenstr.*, 1936, LIV, 315-321, "Atelektase und Pleuritis," *Wien. Klin. Wochschr.*, 1936, XLIX, 1092.
- LENN, R., "Pleural Effusions," *Wien. Klin. Wochschr.*, 1924, XXXVII, 1206-1209; "Zur Röntgendiagnose der Bronchuskarzinome," *Fortschr. Röntgenstr.*, 1926, XXXIV, 4, 455, "Weiter Beiträge zur Röntgendiagnose der Bronchuskarzinome," *Fortschr. Röntgenstr.*, XXXVI, 2, 305, "Die Röntgendiagnostik der intrathorakalen Tumoren und ihre Differentialdiagnose," Julius Springer, Wien, 1929, "Mediastinal Jerkung, a Functional Symptom in Low-grade Bronchostenoses," *Fortschr. Röntgenstr.*, Jan., 1933, abstr. *Amer. Year Book*, 1934, 145.
- LESLIE, M., and BELL, W. S., "Malignant Thymoma, a Case," *Brit. Med. Journ.*, Dec. 3, 1932.
- LEVITEN, J., and BRUNN, H., "Study of Roentgen Appearances of the Lobes of the Lung and Interlobar Fissures," *Radiology*, Dec., 1935, XXV, 6.
- LEVY, C. S., "Congenital Absence of One Lung," *Amer. Journ. Med. Sci.*, 1920, CLIX, 237-246.
- LEVY, H., "Atypical Roentgen Appearance of Pulmonary Infarction in Patients with Heart Failure," *Amer. Journ. Roentgenol.*, 1936, XXXV, 635-639.
- LEWALD, L. T., "Subphrenic Abscess and its Differential Diagnosis Roentgenologically Considered," *Arch. Surg.*, 1923, X, 544-556, "New Growths of the Lung," *Radiology*, March, 1925.
- LICHTENSTEIN, H., "Cavities in the Lung in Lymphogranulomatosis," *Zeitschr. Tbl.*, 1932, LXIV, H. 6, 429.
- LIEBMANN, E., "Miliary Amyloidosis," in SCHINZ "Lehrbuch d. Röntgendiagnostik," 1932, II, 797; "Miliary Tuberculosis," *ibid.*, 834, "Dermoid Cyst," *ibid.* 1114.
- LOCKWOOD, A. L., "Abscess of the Lung," *Surg. Gyn. Obst.*, 1922, XXXV, 461.
- LOESCHKE, H., "Die Morphologie des normalen und emphysematösen Acinus der Lunge," *Beitr. z. Path. Anat.*, 1921, LXVIII, 213-223.

- LOESCHKE, H., and DEHOFF, E., "Pathologische Anatomie des Lungenapitzentuberkulose." *Ergebn. d. ges. Tuberk.*, 1931, II, 81-124.
- LORD, F. T., "Certain Aspects of Pulmonary Abscess, from an Analysis of 227 Cases." *Boston Med. Surg. Journ.*, 1925, CXCII, 1, 785.
- LUDIN, M., "Des solitaire, umschriebene, rundliche Schatten im Lungenröntgenogramm," *Fortschr. Röntgenstr.*, 1926, XXXIV, 899-904: "Aneurysma der Arteria Pulmonalis." *Acta. Rad.*, 1933, XIV, 259-261.
- MCDONALD, S., "Pneumoconiosis," *Brit. Med. Journ.*, 1927, II, 1025.
- MCDUGALL, J. B., "The Tomograph, its Uses in Pulmonary Tuberculosis," *Lancet*, July 25, 1936, 185.
- MCDUGALL, J. B., and CRAWFORD, J. H., "Tomography," *Amer. Rev. Tuberc.*, 1937, XXXVI, 163.
- MCGHEE, W. H., "Importance of Roentgen Examination in Polypoid Tumours of the Larynx," *Amer. Journ. Roentgenol.*, 1933, XXX, 464.
- MCPHEDRAN, F. M., "Diagnosis and Classification of Pulmonary Tuberculosis in Childhood and Adolescence," *Amer. Rev. Tuberc.*, 1929, XX, 532.
- MAINZER, F., "Über isolierte Lungenbilharziose, besonders im Röntgenbild, und ihre Differential diagnose gegenüber der Lungentuberkulose," *Fortschr. Röntgenstr.*, Aug., 1936, LIV, 154-173.
- MAINZER, F., and YALOUSIS, E., "Über latente Lungenkrankung bei manifester Blasenbilharziose (Nach Röntgenuntersuchungen)," *Fortschr. Röntgenstr.*, Oct., 1936, LIV, 373-381.
- MANGES, W. F., "X-ray Diagnosis of Non-opaque Foreign Bodies in the Air Passages," *Amer. Journ. Roentgenol.*, 1922, IX, 288; "Primary Carcinoma of the Lung: Roentgendagnosis and Treatment," *ibid.*, June, 1932; "Non-opaque Foreign Bodies in the Air Passages," *Brit. Journ. Rad.*, April, 1926, XXXI, No. 309, 119; Paper read before Intern. Nat. Cong. Rad., 1925; "Atelectasis as a Roentgen Ray Sign of Foreign Body in the Air Passages," *Amer. Journ. Roentgenol.*, 1934, II, 517.
- MANGES, W. F., and FARRELL, J. T., "Atelectasis," *Amer. Journ. Roentgenol.*, Oct. 30, 1933, 429.
- MARET, P. J., "Differential Diagnosis of Sputum in Chronic Lung Infections," *Brit. Med. Journ.*, May 4, 1929, 809; "A Note on Chronic Infections of the Lungs," *Lancet*, March 5, 1932, 511; "A Note on Monilia Infection of the Respiratory Tract," *Brit. Med. Journ.*, May 27, 1933, 917.
- MATROGORDATO, A., "Studies in Experimental Silicosis and other Pneumoconioses," *Publicn. S. African Inst. Med. Res.*, March 31, 1922, No. 15; "Contribution to the Study of Miners' Phthisis," *Publicn. S. African Inst. Med. Res.*, Dec., 1926.
- MAXIMOW, ALEX. A., and BLOOM, WM., "A Textbook of Histology," W. B. Saunders & Co., London, 1935.
- MAYLARD, A. E., "Abnormalities of the Lobes of the Human Lung," *Journ. Anat. and Phys.*, 1885, XX, 34-38.
- MED. RESEARCH COUNCIL REPORT, "Effects of Alumina Dust," *Brit. Med. Journ.*, Dec. 19, 1936, 1273.
- MEIKLEJOHN, A., cit. BROMLEY, J., *Brit. Journ. Rad.*, 1934, VII, 271.
- MEREWETHER, E. R. A., "Pulmonary Fibrosis in Asbestos Workers," *Journ. Indust. Hygiene*, June, 1930, XII, No. 6.
- MILLER, E. D., "Emphysema," *Amer. Rev. Tuberc.*, Sept., 1933, XXVIII, 359-369.
- MILLER, W. S., "Tuberculous Lung in which Large Emphysematous Bulla was mistaken for Cavity," *Amer. Rev. Tuberc.*, Sept., 1933, XXVIII, 359-369; "Key Points in the Lung Structure," *Radiology*, March, 1935, IV, 3, 173-177; "The Lung," C. C. Thomas, Illinois, 1937.
- MINTZ, N., "Fibroma of Pleura," *Journ. Mt. Sinai Hosp.*, 1935, II, 38-43.
- MISSKE, B., and SYLLA, A., "Röntgenologische Studien des Lungenbildes bei Grippekranken der Epidemie, 1928-29," *Fortschr. Röntgenstr.*, July, 1930, XLII, 4-18.
- MOIR, P. J., "Dermoid Cysts and Teratomata of the Mediastinum," *Brit. Med. Journ.*, March 7, 1936, 463.

- MÖLLER, FLEMMING, "The Radiographic Picture in Chalcosis, and its Differential Diagnosis from Other Affections of the Lungs," *Acta Rad.*, 1927, VIII, 193-208; "Cystic Disease of the Lung," *ibid.*, 1928, IX, 460; "Silicosis in Porcelain Workers, from the Roentgenological and Clinical Points of View," *ibid.*, 1934, XV, 587-602.
- MONIZ, E., and CARVALHO, LOPO DE, "Visibility of the Pulmonary Vessels," *Acta Rad.*, 1933, XIV, 5, No. 81, 433-451.
- MONIZ, E., CARVALHO, LOPO DE, and ALMEIDA LIMA, "L'angiopneumographie et son application dans la Tuberculose Pulmonaire," *La Presse Médicale*, du 13 Juillet, 1932, No. 56.
- MORLAND, A., "Formation and Treatment of Cavities in the Lung," *Lancet*, Dec. 1937, 1311.
- MORLOCK, H. V., and SCOTT PINCHIN, J., "Tracheal Diverticula," *Lancet*, July, 1933, II, 236; "Lung Abscesses and their Treatment," *ibid.*, June 15, 1935, 1369-1373; "Benign Neoplasm of the Bronchus," *Brit. Med. Journ.*, Aug. 24, 1933.
- MORRIS, HUGH, "An Unusual Intrathoracic Lesion," *Brit. Journ. Rad.*, Aug., 1933, VI, No. 68.
- MORTON, S. A., "Roentgen Findings in Pick's Disease," *Radiology*, 1936, XXVII, 619-621.
- NAVEAU, P., and PESQUE, M., "Massive Atelectasis in Pulmonary Tuberculosis," *Arch. Méd. Chir. de l'Appareil Respiratoire*, 1934, IX, 239; also *Brit. Med. Journ.*, Dec., 1934, 81.
- NEIL, J. HARDY, and OTHERS, "Anatomy of the Bronchial Tree and its Clinical Application," *Annals of Otology, Rhinology and Laryngology*, June, 1937, XLVI, 2, 338.
- NELSON, H. P., "Irradiation of Tracheobronchial Lymphatic Glands," *Lancet*, Nov. 22, 1930, 118; "Postural Drainage of the Lungs," *Brit. Med. Journ.*, Aug. 11, 1934, 251-253.
- NELSON, R. L., "Congenital Cystic Disease of the Lung," *Journ. Paediat.*, 1932, I, 233-238.
- NEUMAN, "Klinik der beginnender Tuberkulose Erwachsener," Julius Springer, Wien, 1924.
- Ogilvie, W. H., "Recent Advances in Surgery," Churchill, Ltd., 2nd edn., 1929.
- OPPE, E. L., "First Infection of Tuberculosis by Way of the Lung," *Amer. Rev. Tuberc.*, Nov., 1920, IV, 9; "Pathology of Tuberculosis in Childhood, and its Bearing on Clinical Work," *Brit. Med. Journ.*, Dec., 1927, 1130.
- OPPENHEIMER, E. H., "Experimental Studies on the Pathogenesis of Epituberculosis," *Bull. Johns Hopkins Hosp.*, 1935, LVII, 247.
- OSLER, SIR WM. (and McCRAE, T.), "The Principles and Practice of Medicine," London and New York, D. Appleton & Co., 1923.
- OTTEV, M., "Die Röntgendiagnose der Lungengeschwülste," *Fortschr. Röntgenstr.*, 1910, XV, 1-31.
- OWEN, cit. GRÄBERGER, *Acta Rad.*, 1931, XII, 240-253.
- PANCOAST, H. K., and PENDERGRASS, E. P., "The Upper Respiratory Tract in Children," *Amer. Journ. Roentgenol.*, 1930, XXIII, 258; "Review of Pneumoconiosis," *ibid.*, 1931, XXVI, 4, 556-614; "Superior Pulmonary Sulcus Tumour," *Journ. Amer. Assn.*, Oct. 22, 1932, abstr. *Amer. Year Book of Radiology*, 1933.
- PAPE, R., "Cloudiness of the Lung in Purpura, Multiple Hemorrhages," *Fortschr. Röntgenstr.*, 1933, XLVII, 491.
- PARROT, cit. GHON.
- PARSONS and SPENCE, cit. PARSONS, L. G., "The Childhood Type of Tuberculosis," *Lancet*, May 26, 1934, 1101-1107.
- PASTEUR, W., "Massive Collapse of the Lung," *Brit. Journ. Surg.*, 1914, I, 537-601.
- PEIRCE, C. B., and DIRKSE, P. R., "Pulmonary Pneumatocoele," *Radiology*, 1937, XXVIII, 651-665.
- PEIRCE, C. B., JACOX, H. W., and HILDRETH, R. C., "Roentgen Considerations of Lymphoblastoma," *Amer. Journ. Roentgenol.*, Aug., 1936, XXXVI, 2, 145.

- PENDERGRASS, E. P., and ALLEN, M. K., *Amer. Journ. Roentgenol.*, April, 1934, XXXI, 4, 470-481.
- PENDERGRASS, E. P., and DE LORIMER, A., "Broncholiths and Stone Asthma," *Radiology*, 1935, XXV, 6, 717.
- PETERSON, H. O., "Benign Adenoma of the Bronchus," *Amer. Journ. Roentgenol.*, 1936, XXXVI, 836-843.
- PFAHLER, G. E., "Roentgendiagnosis of Mediastinal Tumours and their Differentiation," *Amer. Journ. Roentgenol.*, April, 1934, XXXI, 4, 458-469.
- PHEMISTER, D. B., STEEN, W., and VOLDERAUER, J. C., "Roentgenological Investigation of Dermoid Cyst," *Amer. Journ. Roentgenol.*, July, 1936, XXXVI, 1, 14.
- PILGER, W., "Four Cases of Healed Miliary Tuberculosis of Lungs," *Roentgenpraxis*, April 15, 1932, IV, 334; *Amer. Year Book*, 1934, 158.
- PILOT, I., "Mesenchymatous Tumours of Lung and Pleura," *Radiology*, 1930, XIV, 391-400.
- POHL, R., "Der Lobus posterior der Lunge," *Fortschr. Röntgenstr.*, Nov., 1932, XLVI, 583-587.
- POIRIER, P., and CHARPY, A., "Traité d'Anatomie Humaine," 2nd edn., IV, Masson et Cie, Paris, 1903.
- POLGÁR, F., "Basal Horizontal Shadows of Lungs," *Roentgenpraxis*, Nov., 1935, VII, 738-742; abstr. *Amer. Year Book of Radiology*, 1936, 149.
- PUHL, *Klin. Wochschr.*, 1929, VIII, 1.
- RABIN, C. B., in "Diagnostic Radiology," Ross Golden, Thos. Nelson, 1936, 90.
- RANKE, K. E., "Primäraffekt, sekundäre und tertiäre Stadien der Lungentuberkulose," *Deutsch. Arch. f. Klin. Med.*, 1916, CXIX, 201.
- REDEKER, F., "Über die Fruhinfiltrate und die Irrlehre von gesetzmässigen Zusammenhang der sogenannten Spitzentuberkulose mit der Erwachsenenphthise," *Deutsch. Med. Wochschr.*, Jan. 14, 1927, No. 3, 97-99; "Die haematogenen Streuformen die kindlichen Lungentbk.," "Handbuch der R. diagnostik," St. Engel and Schall, 1933, 307.
- RICHARDS, G. E., "Interpretation of Triangular Basal Shadows on Radiograms of the Chest," *Amer. Journ. Roentgenol.*, 1933, XXX, 3, 289.
- RIGLER, L. G., and ERICKSEN, L. G., "Inferior Accessory Lobe of the Lung," *Amer. Journ. Roentgenol.*, 1933, XXIX, 385-392.
- ROBINSON, A. A., "Congenital Cystic Lung," *Brit. Med. Journ.*, Nov., 1935, 837.
- ROBINSON, W. W., "Pulmonary Syphilis in Adults," *Radiology*, Nov., 1935, XXV, 5, 596-599.
- ROSTOSKI, SAUPE, and SCHMORL, "Die Bergkrankheit der Erzbergleute in Schneeberg in Sachsen," *Leitschr. f. Krebsforschung*, 1926, XXIII, 360.
- RUCKENSTEINER, E., and HÖRTNAGL, H., "Über einen seltenen Fall von offener Wabenlunge," *Fortschr. Röntgenstr.*, Dec., 1934, L, 6, 571.
- SALINGER, H., "Die Knochenbildung in d. Lunge mit besond. Berücksicht d. Tuberös. Form.," *Fortschr. Röntgenstr.*, 1932, XLVI, 269.
- SAMSON, K., and ENGEL, St., "Lungerkrankungen," in "Handbuch d. R. diagnostik im Kindesalter," G. Thieme, Leipzig, 1933.
- SAMPSON, H. L., HEISE, F. H., and BROWN, L., "A Study of Pulmonary and Pleural Annular Radiographic Shadows," *Nat. Tuberc. Ass. Tr.*, New York, 1918, XIV, 171-180.
- SANTE, L. R., "Massive Collapse of the Lung," *Radiology*, 1927, VIII, 1-13; "The Chest," *Annals of Roentgenology*, XI, Paul Hoeber, New York, 1931.
- SAPHIR, cit. KARSNER (1935), 600.
- SAUERBRUCH, F., "Die Chirurgie der Brustorgane," J. Springer, Berlin, 1928, 869.
- SAUL, W., "Bronchitisformen in Röntgenbilde," *Fortschr. Röntgenstr.*, 1930, XLII, 2, 223.
- SAYERS, R. R., and MERIWETHER, F. V., "Miliary Lung Disease of Unknown Cause," *Amer. Journ. Roentgenol.*, Mar., 1932.
- SCHAFFNER, "Über den Lob. access. inf d. Mensch. Lunge," *Virchow's Arch.*, 1898, 152.
- SCHALL, L., and HOFFMANN, F., "Zur Anatomie der Interlobärsalten," *Fortschr. Röntgenstr.*, Dec., 1930, XLII, 714-729.

- SCHENK, S. G., "Congenital Cystic Disease of the Lungs," *Amer. Journ. Roentgenol.*, 1936, XXXV, 604-629.
- SCHINZ, H. R., BAENSCHE, W., and FRIEDL, E., "Lehrbuch der Röntgendiagnostik," Geo Thieme, Leipzig, 1932.
- SCHINZ, H. R., and GASSER, E., "Mediastinallipom," *Röntgenpraxis*, Nov., 1933, V, 821-823.
- SCHLEIER, F. J., "Dermoid Cyst of the Pleural Cavity," *Journ. Amer. Med. Ass.*, 1923, LXXXIV, 1038.
- SCHMOLL, G., and JUNGHANS, H., "Die gesunde und kranke Wirbelsäule im Röntgenbild," Geo. Thieme, Leipzig, 1932.
- SCHOBEL, O., and SELLARDS, A. W., *Philippine Journ. Sci.*, 1926, XXXI, 1; cit. "Recent Advances in Pathology," Hadfield and Garrod, Churchill, Ltd., 1934.
- SCHONFELD, H., "Beiträge zur Röntgendiagnostik pleuritischer Prozesse im Kindesalter," *Monatschr. für Kinderheilk.*, 1928, XXXIX, 385-471.
- SCHWARZWANN, A., "Über generalisierte carcinomatöse Lymphangitis der Lungen," *Acta Rad.*, 1934, XV, 491-501.
- SCRINGER, F. A. C., "Post-operative Massive Collapse," *Journ. Surg. Gyn. and Obst.*, 1921, XXXII, 486-492.
- SGALIZER, M., and STOUR, W., "Zur Röntgenuntersuchung d. Luftrohre," *Fortschr. Röntgenstr.*, 1924, XXII.
- SHULL, J. R., "Asbestosis, a Roentgenologic review of 71 Cases," *Radiology*, 1936, XXVII, 279-292.
- SICARD, J. A., and FORESTIER, J., "Exploration Radiologique par l'Huile iodée," *Presse Médicale*, 1923, XLIV, 'The Use of Lipiodol in Diagnosis and Treatment,' Oxford University Press, New York and London, 1932.
- SIMONDS, M., "Ueber verästelte Knochenbildungen der Lunge," *Fortschr. Röntgenstr.*, 1918, XXV, 393-401.
- SIMON, S., "Röntgenologischer Nachweis des primären Lungenherdes bei der Bronchialdrüsen tuberkulose," *Beitr. Klin. Tbk.*, XXVI; "Das Bild der Lungentuberkulose des Kindes," in St. Engel and Schall, "Handbuch d. R. diagnostik im Kindesalter," 350.
- SMITH, F. W., and STRACHAN, A. S., "Lymphoid Tissue in the Lung in a Case of Status Lymphaticus," *Publ. of S. A. Inst. for Med. Research*, 1932.
- SLUKA, E., "Die Hilustuberkulose des Kindes im Röntgenbild," *Wien. Klin. Wochschr.*, 1912, XXV, 259.
- SMITH, A. R., "Silicosis among Rock-drillers, Blasters, and Excavators in New York City," *Journ. Indust. Hygiene*, 1929, II, 39-60.
- SMITH, C. H., "Tuberculosis of Childhood," *Ann. Int. Med.*, Mar., 1935, VIII, 1090-1120.
- SMITH, S., "Congenital Cystic Disease of the Lung," *Brit. Med. Journ.*, 1925, I, 1003.
- SOKOL, L., "Massive Atelectasis complicating Pulmonary Tuberculosis," *Amer. Rev. Tuberc.*, Oct., 1932.
- SFALTENHOLTZ, WERWER, "Hand atlas of Human Anatomy," III, Lippincott & Co., London.
- SPARKS, J. V., "Pulmonary Asbestosis," *Radiology*, Dec., 1931.
- STAUB-OETIKER, H., "Die Pneumokoniose der Metallschleifer," *Deutsch. Arch. Klin. Med.*, 1916, CXIX, 469-481.
- STEINER, P. E., and FRANCES, B. F., "Primary Apical Lung Carcinoma," *Amer. Journ. Cancer*, 1934, XXII, 776-785.
- STEWART, H. L., KENNEDY, P. J., and JAMES, A. E., "Congenital Cyst of the Lung," *Arch. Path.*, 1932, XIV, 627-636.
- STEWART, M., and FALLIS, "The Pulmonary Fibrosis of Hematite Miners," *Journ. Path. and Bact.*, 1934, XXXIV.
- STEWART, W. H., and ILLICK, H. E., "Where is the Diaphragm?" *Radiology*, 1934, XXII, 6.
- STIEVELMANN, cit. WESTERMARK, N., *Acta Rad.*, 1933, XVI, 542.
- STOERK, O., "Über angeb. bläsige Missbildungen der Lunge," *Wien. Med. Wochschr.*, 1897, X, 25.

- SUTHERLAND, C. L., and BRYSON, S., "Report on the Incidence of Silicosis in the Pottery Industry," H.M. Stationery Office, 1926; and "Report on Silicosis among Sandstone Workers," H.M. Stationery Office, 1929.
- TAYLOR, H. K., and BORROWITZ, I. D., "Fibrin Bodies in Artificial Pneumothorax," *Radiology*, 1935, XXV, 274-284.
- TEALL, C. G., "Radiological Aspects of Bronchiectasis in Children," *Brit. Journ. Rad.*, 1937, X, 637.
- TERRELL, E. E., ROBERTSON, O. H., and COGGESHALL, L. T., "Experimental Pneumococcus Lobar Pneumonia in the Dog," *Journ. Clin. Investigation*, 1933, XII, 393.
- THOMAS, cit. ASSMANN, "Anatomisch-physiologische Grundlagen der Bogenunterteilung des Zwerchfells im Röntgenbilde," *Deutsch. Med. Wochschr.*, 1921; also *Fortschr. Röntgenstr.*, 1923, XXXI.
- TILLIER, R., "Diagnostic Radiologique des Kystes Hydatiques suppurés du Poumon," *Bull. et Mém. Soc. de Radiol. Méd. de France*, June, 1932, XX, 361-368.
- TILLIER, R., and LE GENISSEL, M., "Radiology of Hydatid Cysts," *Journ. de Radiologie et d'Electrologie*, May, 1936, XX, 273-302.
- TURNBULL, cit. HADFIELD and GARROD.
- TWINING, E. W., "Radiology of the Chest," *Brit. Journ. Rad.*, 1931, IV, 658; "Tomography, by Means of a Simple Attachment to the Potter-Bucky Couch," *Brit. Journ. Rad.*, April, 1937, X, 112; "Lateral View of the Lung Apices by Interclavicular Projection," *ibid.*, 123.
- UDE, W. H., "Roentgenologic Studies in Early Lobar Pneumonia," *Amer. Journ. Roentgenol.*, 1931, XXVI, 5, 691.
- UDVARDY, L., "Durchwanderungsexsudate der Pleura und Herdpneumonien bei Bauchhöhlenprozessen," *Röntgenpraxis*, 1934, VI, 785-792.
- VALLEBONA, A., "Technique of Roentgenographic Disassociation of Shadows," *Radiol. Med.*, 1930, XVIII, 1090; *Radiology*, 1931, XVII, 340.
- VAN DER MANDELE, "Über einen Fall von Pleuritis Mediastinalis und über d. Doppelkontur des Herzschattens," *Fortschr. Röntgenstr.*, 1926, XXXIV, 84-97.
- VAN ZWALUWENBURG, "Apical Pleuritis," *Amer. Rev. Tuberc.*, 1922, VI, 106.
- WAINWRIGHT, J. M., and NICHOLS, H. J., "The Relation between Anthracosis and Pulmonary Tuberculosis," *Amer. Journ. Med. Sci.*, 1905, CXXX, 386-399.
- WARFIELD, C. H., "Roentgen Diagnosis of Aneurysms of Innominate Artery," *Amer. Journ. Roentgenol.*, 1935, XXXIII, 350-358.
- WARNUNG, BJORN, "Calcification of the Larynx," *Acta. Rad.*, 1934, XV, 83, 8.
- WASSON, W. W., "Radiography of the Infant Chest," *Radiology*, 1925, V, 365.
- WEIL, A., "Drei Fälle von Lungentumoren mit ungewöhnlichen röntgenologischem Befund," *Fortschr. a. d. Geb. d. Röntgenstrahlen*, 1912, XIX, 142-148.
- WEINGÄRTNER, cit. ASSMANN, *Diagnostik Inn. Krankheiten*, 1934, I, 227.
- WEISS, T., and BIERMANN, E., "Intratracheal Tumour diagnosed Radiologically and Removed," *Röntgenpraxis*, April 1, 1932, abstr. *Amer. Year Book*, 1932, 201.
- WESSLER, H., and JACHES, L., "Clinical Roentgenology of Diseases of the Chest," Southworth & Co., New York City, 1923.
- WESTERMARK, N., "The Situation of Pleural Exudate in Obstructive Atelectasis of the Lung," *Acta. Rad.*, 1935, XVI, 345; "Entwicklung und Vorkommen von Atelektase bei Lungen-tuberkulose," *Acta. Rad.*, 1935, XVI, 531.
- WHALEN, E. J., "Fungus Infections of the Respiratory Tract," *Arch. Otolarynx*, Oct., 1936, Chicago, 436; abstr. *Brit. Med. Journ.*, Jan. 23, 1937, 15.
- WHITBY, L. E. H., "Aspergillosis," *Brit. Ency. of Med. Practice*, Rolleston, 1936, II, 165, Butterworth, London.
- WHITE, J. DUNCAN, "Abnormalities of the Bony Thorax," *Brit. Journ. Rad.*, 1929, II, 351.
- WIGBY, P. E., and SANDERS, C. B., "Syphilitic Gumma of the Lung," *Radiology*, Nov., 1935, XXV, 629.
- WELLS, H. S., "The Pulmonary Effect of over Three Years' Exposure of Guinea-pigs to Silica Dust," *Amer. Rev. Tuberc.*, 1928, XVII, 253-287.
- WILLIS, R. A., "Spread of Tumours in the Human Body," J. & A. Churchill, Ltd., 1934.

- WILMS, M, "Ueber die Dermoidcysten und Teratome, mit besonderer Berücksichtigung des Dermoids der Ovarien," *Deutsch. Arch. Klin. Med.*, 1893, LV, 289-398.
- WINKLER, cit. WILLIS, R. A., "Spread of Tumours in the Human Body," J. & A. Churchill, Ltd., London, 1934.
- WOLFF, A. O., "Contribution à la topographie des ganglions intrathoraciques," *Acta. Rad.*, 1935, XVI, 674.
- WOOD, H. C., "A Frame for holding Infants Upright during Roentgenography," *Amer. Journ. Roentgenol.*, 1934, XXXI, 396.
- WOOD, W. BURTON, "Pulmonary Asbestosis," *Brit. Journ. Rad.*, May, 1934, VII, 77, 277.
- WOOD, W. BURTON, and GLOYN, S. R., "Pulmonary Asbestosis complicated by Pulmonary Tuberculosis," *Lancet*, 1931, II, 954.
- ZELDUES DES PLANTES, B. G., "New Method of Differentiation in Radiography (Planigraphy)," *Acta Rad.*, 1932, XIII, 182-191; "Planigraphy," Kemink & Son, Utrecht, 1934.
- ZIGGLER, K., "Die Lymphogranulomatose" (Hodgkin's Disease). *Erg. Inn. Med.*, 1927, XXXII, 46.
- ZUFFINGER, A., "Röntgen Examination of Pharyngeal Tumours," *Fortschr. Röntgenstr.*, 1935, LII, 468-480.
- ZWIFEL, C., "Der Zwerchfellhochstand beim Lungeninfarkt," *Fortschr. a. d. Geb. Röntgenstrahlen*, Sept., 1935, LII, 222-227.

CHAPTER XXXVI

THE X-RAY APPEARANCES OF THE LUNGS FOLLOWING PNEUMOTHORAX AND SURGICAL PROCEDURES

By PETER KERLEY, M.D., F.R.C.P. D.M.R.E.

ARTIFICIAL PNEUMOTHORAX

RECENT STATISTICS from practically all countries show that the prognosis in pulmonary tuberculosis has vastly improved during the last twenty-five years.

This improvement is due to a number of factors, such as earlier diagnosis and improved conditions of living, but chiefly to the successful use of artificial collapse of the lung.

The indication for active treatment in a case of pulmonary tuberculosis is chronic cavitation. The patient's general condition, his occupation, and his economic position are also important considerations outside the radiologist's sphere. Not all cavities demand active interference, and it is now well known that fresh thin-walled cavities developing rapidly in a zone of exudative infiltration can be healed by rest and medical treatment. A thick-walled cavity surrounded by fibrous tissue

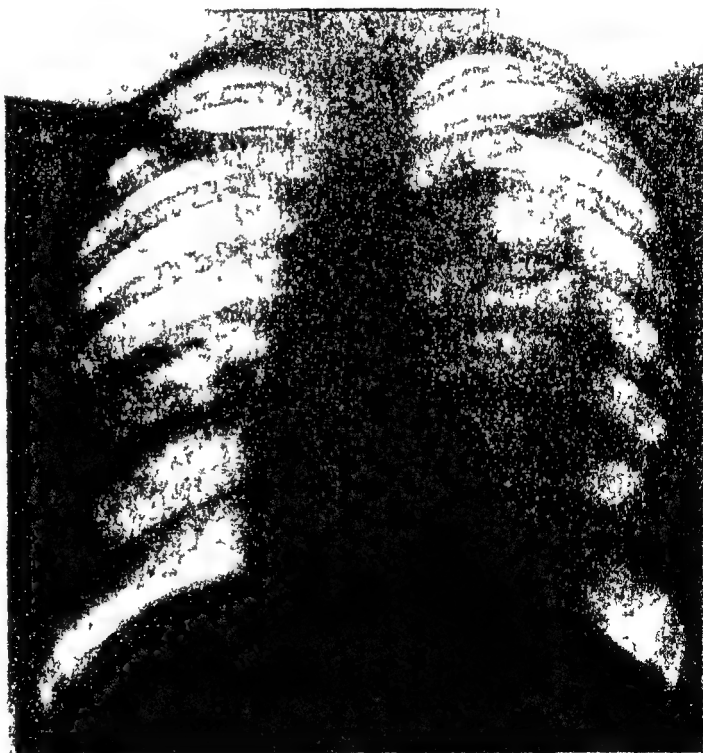


FIG 317.—Left artificial pneumothorax. There is a thick adhesion between the 3rd rib and the lung surface over the cavity. The bronchus draining the cavity is clearly visible. Unsatisfactory collapse with inhalation infection of the right middle and lower lobes.

will not heal spontaneously, and the patient's outlook depends largely on whether such a cavity can be collapsed or not. It is obvious that extensive

progressive infiltration in the opposite lung is a contraindication to pneumothorax treatment except in a small group of cases where the disease in both lungs progresses in spite of rest and gold injections. In such cases a bilateral pneumothorax may be induced.

The X-ray appearances of the lung tissue and pleura surrounding a cavity are no guide at all as to whether the lung can be collapsed successfully or not. Time and again one sees cavities surrounded by dense fibrous tissue and appar-



FIG 318—Right artificial pneumothorax with superimposed atelectasis of the lower lobe. Anterior mediastinal hernia. Moderate hyperemia of the left lung.

ently grossly thickened pleura, and one's impression from the plain film is that this lung will be firmly fixed by pleural adhesions. A pneumothorax is induced and the lung collapses beautifully. The only way by which one can estimate whether a pneumothorax will be successful or not is to induce the pneumothorax.

The old theory of artificial pneumothorax presumed active compression of the lung as the ideal to be aimed at. This theory is now being discarded, and the modern idea is to produce such a condition of affairs in the thorax that the diseased

area of lung tissue will relax and as little pressure as possible be placed on the healthy lung tissue.

At the beginning of a pneumothorax only small quantities of air are introduced into the pleural cavity. On the radiogram the edge of the lung becomes clearly visible, and in most cases cavities in the lung also show up much better. As the pressure is increased by refills of air, the lung relaxes more and more towards the mediastinum. The lobar indentations can be seen on its edge and the pulmonary vessels, which are congested as well as compressed, show up with great clarity. During this process cavities in the relaxed lung alter in shape, gradually becoming oval, then elliptical, and finally in successful cases

disappearing. At the same time a fluxionary hyperæmia is taking place in the opposite lung, the vessels becoming thicker and denser, with some consequent loss of aeration. It is possible to confuse engorged end-on vessels in the healthy lung with fresh infiltration, but with good technique the differentiation is easily made.

Mediastinal Movements.—The movements of the mediastinum must be carefully studied during an artificial pneumothorax. In some individuals the mediastinum is a very rigid structure, and in others it is very lax and swings easily to either side. Excessive displacement of the mediastinum frequently occurs during a pneumothorax, and while such displacement is not immediately dangerous it can nullify the benefits of the pneumothorax and predispose to infection of the healthy lung. The X-ray appearances vary according to the rigidity or laxity of the mediastinum.

With a very slack mediastinum the pressure in the pneumothorax may cause a marked displacement of the trachea and mediastinum to the opposite side; this in itself is of little significance until it reaches a stage where it intensifies the fluxionary hyperæmia in the healthy lung. The circulation is then embarrassed, and there is constant danger of infection of the healthy lung. With a rigid mediastinum the heart and trachea do not swing much over to the opposite side, but they rotate considerably with similar embarrassment of the circulation. Unfortunately such circulatory disturbances often develop with a pneumothorax pressure insufficient to cause relaxation of the diseased area of lung tissue.

So-called mediastinal herniæ often develop during pneumothorax in individuals with a very slack mediastinum. In the normal individual there is always a potential space immediately behind the sternum, and under certain circumstances the pleura can slip across this space and appear as a thin semicircular line on the opposite side. Such herniation of the mediastinum is of no significance in a slack mediastinum; it does not signify excessive pressure, and I have often observed its occurrence in patients with partial lower lobe collapse where the relative pressure alterations were trivial. In a rigid mediastinum, however, herniation does not occur unless there is excessive pressure; should herniation occur suddenly, the patient collapses, and indeed the incident may cause death from shock. This is a rare complication, and fortunately radiography reveals the danger early. In the ordinary postero-anterior radiogram the point of potential herniation seen from the front is at the level of the third to the fifth dorsal vertebræ, and this level should be carefully watched. If the heart and trachea are rotated, and if at this level a thin white line appears at all on the contralateral side, there is excessive pressure present.

In many cases excessive mediastinal displacement and/or excessive hyperæmia of the healthy lung develop before there is adequate relaxation of the diseased area. Increasing the pressure with the object of forcibly compressing the cavity does more harm than good and definitely tends to infect the opposite

lung. In some of these cases where the cavity is thin-walled and partially collapsed the pneumothorax need not necessarily be abandoned. Such a cavity may slowly heal, even though its walls do not appear to be in apposition, and, provided clinical evidence shows that the disease is being controlled, the pneumothorax should be maintained at an optimum pressure. If, however, clinical signs point to progression of the disease, the case must be reviewed and

additional or alternative methods of collapse considered.

Pleural Adhesions.—

The commonest cause of inadequate collapse during an artificial pneumothorax is pleural adhesions. Such adhesions can be easily visualised on radio-graphs, they show as opaque bands running from the edge of the lung across the empty pleural space to the ribs. Their thickness cannot be estimated by radiography, nor is it possible to state whether the medial end of an adhesion contains lung tissue or not. The value of radiography is in showing which adhesions are preventing effective collapse.

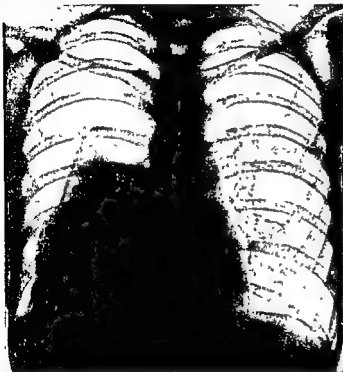


FIG. 319.—Right artificial pneumothorax and superimposed atelectasis of the lung. Small quantity of fluid at the base. Mediastinum not displaced. Healed lesion in the left upper lobe.

Not all adhesions are harmful, and, in fact, some may be beneficial. An apical adhesion, for example, is often an important factor in securing apposition of the walls of a cavity. There are various methods of localising adhesions, but the most effective are undoubtedly stereoscopy and tomography.

There are two methods of dealing with adhesions which are preventing proper collapse. The adhesions may be severed under direct vision through a thoracoscope or the diaphragm may be paralysed. *The division of adhesions by the electro-cautery* is now a simple and painless operation in skilled hands. The operator can see the adhesions, can determine their exact nature, and can even see whether there are tubercles on them or not. In the early days of

pneumolysis, when diathermy was used for severing the adhesions, there was some risk of hæmorrhage and the development of a hæmo- or pyo-pneumothorax. Such complications are now rare, and following the operation one seldom sees anything but a mild degree of surgical emphysema. In properly selected cases the operation is eminently successful in securing complete collapse of a cavity.

Phrenic paralysis is often very useful in securing relaxation of adhesions. The diaphragm may be paralysed temporarily by simply crushing the phrenic nerve, or it may be paralysed permanently by avulsing the nerve. The modern practice is first to crush the nerve, and if the result is obviously good, avulsion is carried out later. The characteristic X-ray appearances of diaphragmatic paralysis develop in a week to ten days after the operation. The diaphragm slowly rises to the cadaveric position, and its movements either disappear or become paradoxical. As a result of the altered position of the diaphragm there is increased relaxation of the lung, while the capacity of the pleural space is decreased and adhesions tend to become slack. In certain cases it may be necessary to perform both phrenic avulsion and pneumothorax before the lung is properly collapsed.

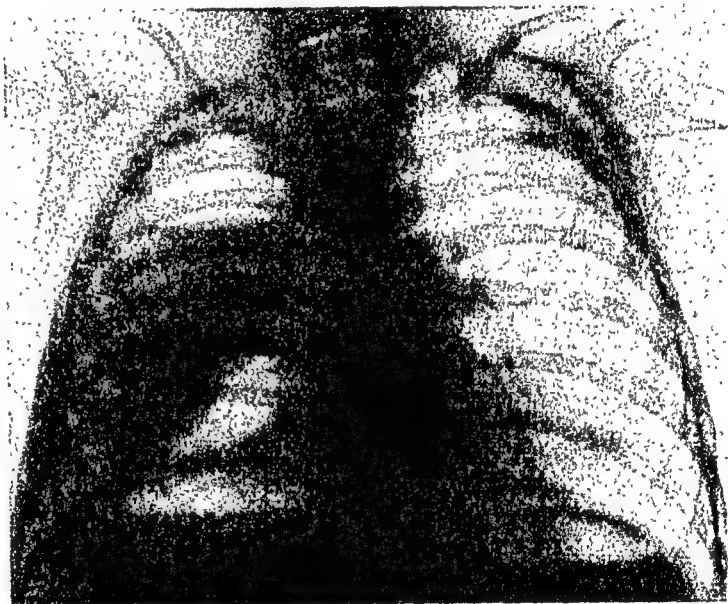


FIG. 320.—Right artificial pneumothorax complicated by fluid. The lower lobe has re-expanded and pushed the hydro-pneumothorax upwards.

Selective Pneumothorax.—In very many cases in which a pneumothorax is induced only one lobe is diseased. Considerable work has been done with the object of discovering a method whereby only the diseased area is collapsed. In an ordinary pneumothorax the whole of the lung, including both healthy and diseased areas, collapses. This does not predispose to infection of the healthy area, but it is obviously to the patient's advantage to keep as much healthy lung tissue as possible inflated. *Maurer* has recently devised a method which promises to be effective in securing selective collapse of a diseased area. A pneumothorax is induced and the whole of the lung collapses. The natural

tendency of the lung is to re-expand unless air refills are carried out at regular intervals. *Maurer* introduces into the pleural cavity small quantities of a solution of glucose. This substance provokes a fibrinous pleuritis, and by skilfully alternating the air injections and the glucose injections the healthy area is allowed to re-expand and stick while the diseased area is kept compressed. A distinct advantage of this method is that the mediastinal leaves

of the pleura become adherent and a slack mediastinum is converted into a rigid one.

Oleothorax. — In most cases of artificial pneumothorax with a free pleura there is no difficulty in keeping the lung relaxed by frequent refills of air, and as a rule the intervals between the refills gradually become longer. Occasionally one sees a case in which the lung tends to re-expand so rapidly that refills at least two or three times a week would be necessary to keep it relaxed. It was thought that if some heavy substance, such as oil, were injected into the pleural

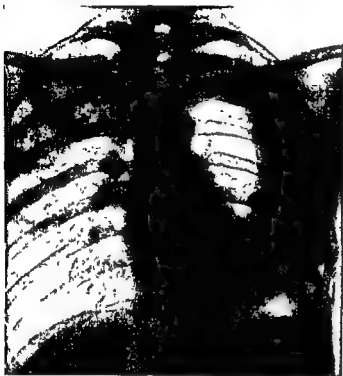


FIG. 321 —Pleural obliteration on the left side. Fresh infection of the right lung.

cavity, the weight of this substance would keep the lung down and eliminate the necessity for frequent air refills. Various oils were employed, and the one which gained most favour was a French compound known as Gomenol. There was no doubt whatever of the effectiveness of a heavy oil in the pleural cavity in keeping the lung compressed, but unfortunately a large number of the patients so treated eventually coughed up the oil—in other words, the lung ruptured. The reason for this complication is obscure—but probably the oil causes active compression instead of relaxation of the lung. The method has been almost completely abandoned because of the danger of rupture, but it was undoubtedly successful in a small percentage of cases.

The appearances of an oil injection of the pleural cavity are identical with those of an encysted pleural effusion, i.e. a dense, well-defined opacity with no markings visible through it and widening of the interspaces over it.

Hydropneumothorax.—Fluid in the pleural cavity is a frequent complication of artificial pneumothorax. It may develop insidiously without known cause, but excessive exercise by the patient certainly tends to provoke it.

Fluid in a pneumothorax finds its own level, and with the patient erect appears as a dense opacity with an upper horizontal level. The level varies with the position of the patient, who is usually aware of the presence of the fluid. A small hydrothorax is not of much significance, and with care and suitable treatment it often resolves. Sometimes the fluid accumulates rapidly and may so increase the intrapleural pressure that respiration and circulation are embarrassed. Aspiration is then necessary. The X-ray appearances of excessive pressure on the mediastinum and healthy lung are an obvious guide to the time for aspiration.

In many cases complicated by fluid it is difficult to maintain the intrapleural pressure at an optimum level, and the lower lobe tends to re-expand. As the lower lobe expands towards the periphery we see the hydropneumothorax slowly pushed upwards, and when eventually the parietal and visceral pleura over the lower lobe are in apposition, adhesions form and the two layers stick. This may result in a selective collapse of the upper lobe (often

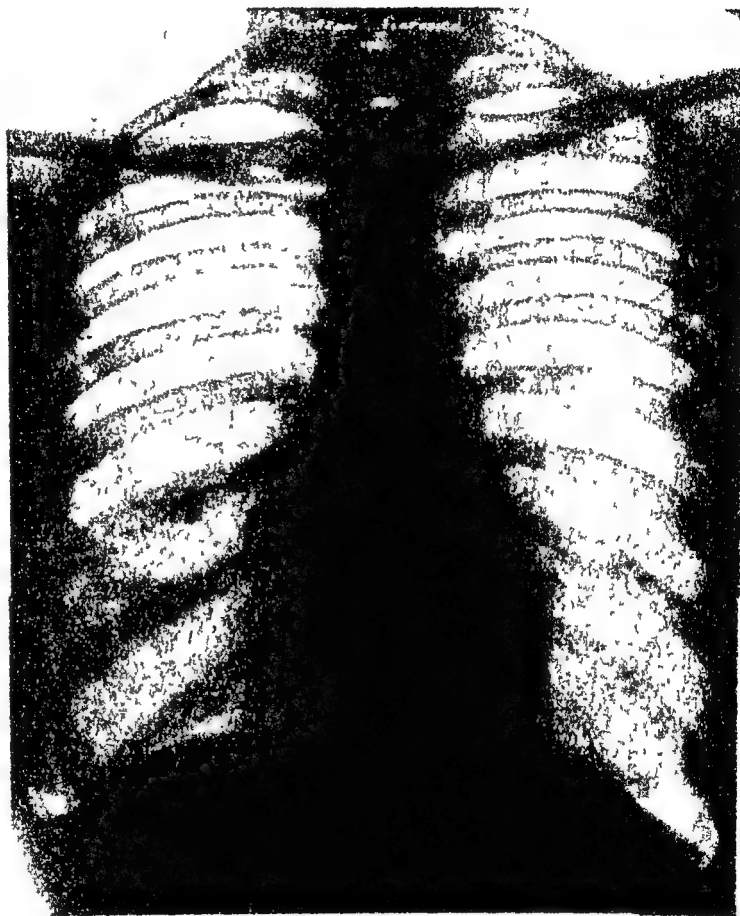


FIG. 322.—Bilateral artificial pneumothorax. Good collapse of both lungs. Small quantity of fluid at the right base.

diseased area), and in a good case the selective collapse can be maintained by judicious refills. If the whole lung re-expands while there is fluid in the pleural cavity, we get a condition known as pleural obliteration. The whole of the affected side shows considerable loss of translucency, and near the periphery there may be no lung tissue visible. Frequently a sharp crescentic line with its convexity outwards separates visible lung tissue from complete opacity. The appearance is similar to that of a resolving pleural effusion, save that in obliteration the intercostal spaces are contracted. Calcification of the pleura is rarely seen after re-expansion of the lung following a hydropneumothorax. Occasionally fluid in a pneumothorax becomes organised into what is known as a fibrin body. Such a body is seen as an oval opacity of homogeneous density lying free in the pleural cavity. Usually a fibrin body can move about freely within the limits of the pneumothorax, but there are a few records of a fibrin body being attached to the parietal pleura by a stalk of fibrous tissue. Calcification of a fibrin body has also been described. Following re-expansion of the lung a fibrin body can be absorbed or remain permanently at the bottom of the pleural cavity. These bodies are of no clinical significance. Occasionally atelectasis develops in a lung partially collapsed by pneumothorax; this is of no clinical significance.

PHRENIC CRUSH AND PHRENIC AVULSION

Temporary or permanent paralysis of the phrenic nerve is usually employed as an accessory method of relaxing adhesions in a pneumothorax. Experience has shown that diaphragmatic paralysis causes more relaxation of the upper lobe than the lower lobe. Although the treatment is viewed with disfavour by many, it has definite value in selected cases. The X-ray appearances of phrenic paralysis are well known. The diaphragm rises one or two interspaces higher than normal and its movements disappear completely or become paradoxical. These phenomena manifest themselves in a week to ten days after the nerve is crushed. Simple crushing of the nerve produces temporary paralysis lasting four or five months, after which period the diaphragmatic movements and position return to normal. Avulsing the nerve causes permanent paralysis, and the affected side of the diaphragm finally becomes a thin fibrous sheath. Occasionally a phrenic crush causes paralysis only for about a month, and rarely it causes permanent paralysis.

The indications for a phrenic operation cannot be discussed here, and indeed can only be assessed in a given case. Roughly speaking, an upper lobe cavity which cannot be collapsed by a pneumothorax and which is unsuitable for thoracoplasty can be helped by phrenic paralysis. Basal cavities are seldom favourably influenced by phrenic paralysis. Should a patient with a phrenic paralysis be subjected to a thoracoplasty, the operative risk is slightly increased, owing to the danger of aspiration pneumonia.

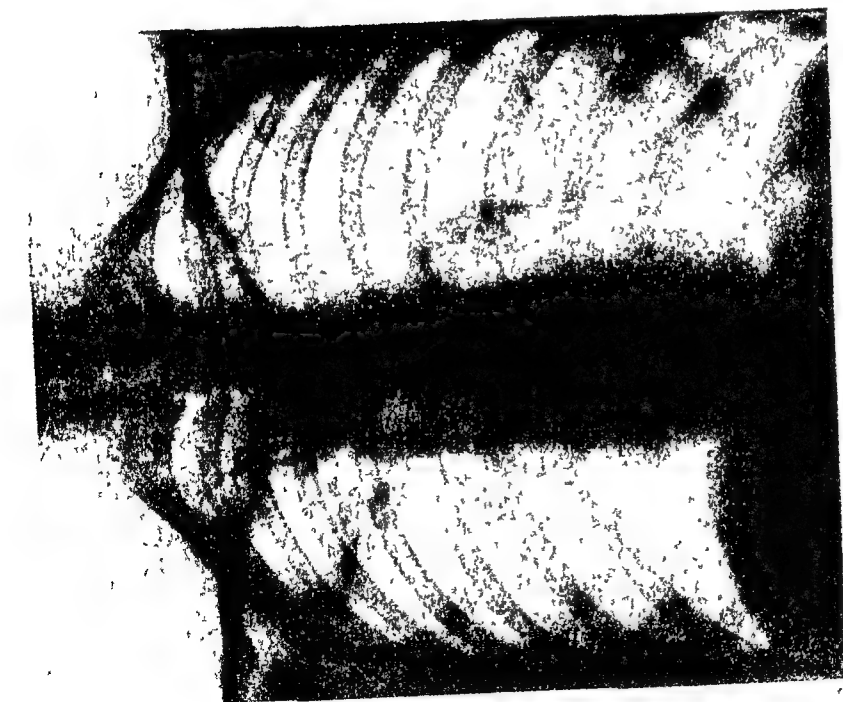


FIG. 323.—Excavation of the right upper lobe. Left lung intact.



FIG. 324.—Same case 8 days after upper seven-rib thoracoplasty. Note the immediate reduction in the size of the cavity, and the oval area of surgical emphysema over the apex.

PLOMBAGE

This is a method of exerting local pressure on a cavity by making a small opening in the chest wall, detaching the lung from the inner surface of the thorax, and filling up the space with wax. Some forms of the wax are radio-opaque and produce a dense shadow, through which nothing can be seen. As



FIG. 325 — Same case as Figs. 323 and 324 one year after thoracoplasty. Clinical cure.

the compression effect of a plombage can only be checked by radiography, the modern tendency is to use a radiotranslucent substance. Plombage has not proved very effective in securing selective collapse of a cavity, but it is often useful in association with other surgical procedures.

EXTRAPLEURAL PNEUMOTHORAX

This is a new method of treatment which has not yet been extensively tried. It promises to be very successful in the treatment of apical cavities with adhesions. The posterior part of the third rib is removed and the parietal pleura over the apex and down to the lung root is

separated so that the upper lobe collapses. The collapse is maintained by an extrapleural pneumothorax until the cavity has healed.

THORACOPLASTY

When all other methods of closing a cavity fail, thoracoplasty must be resorted to. The operation consists in removal of large areas of a number of ribs, so that the whole of the chest in the region of the cavity falls in. With increasing experience and more careful selection of cases, the results of thoracoplasty are steadily improving and the operative mortality is steadily decreasing.

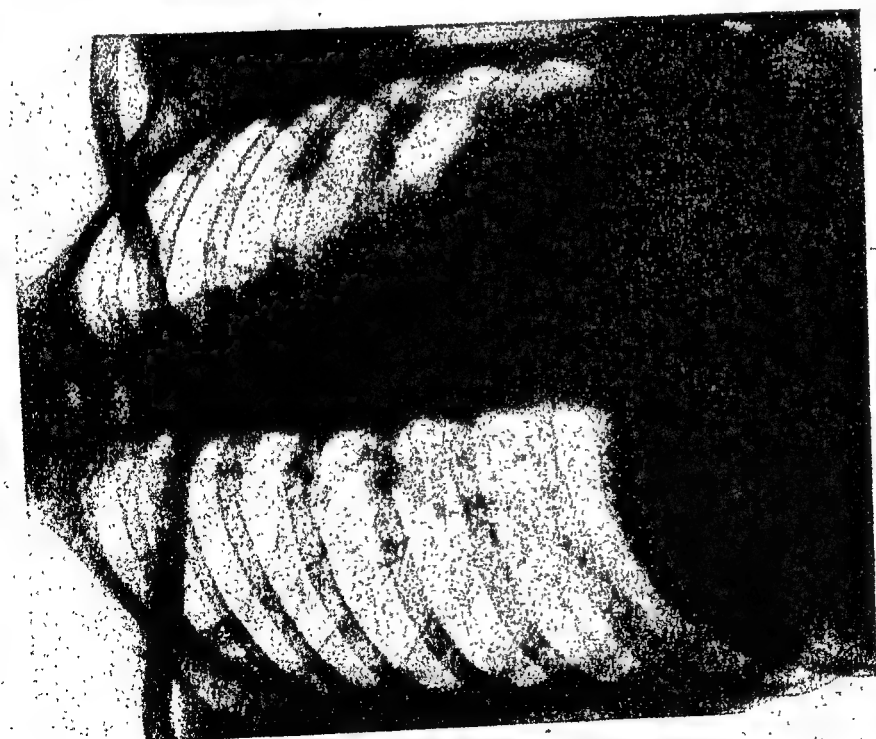


FIG. 326.—Unsuccessful artificial pneumothorax on the left side. Large upper lobe cavities are held open by dense adhesions. Right lung intact.

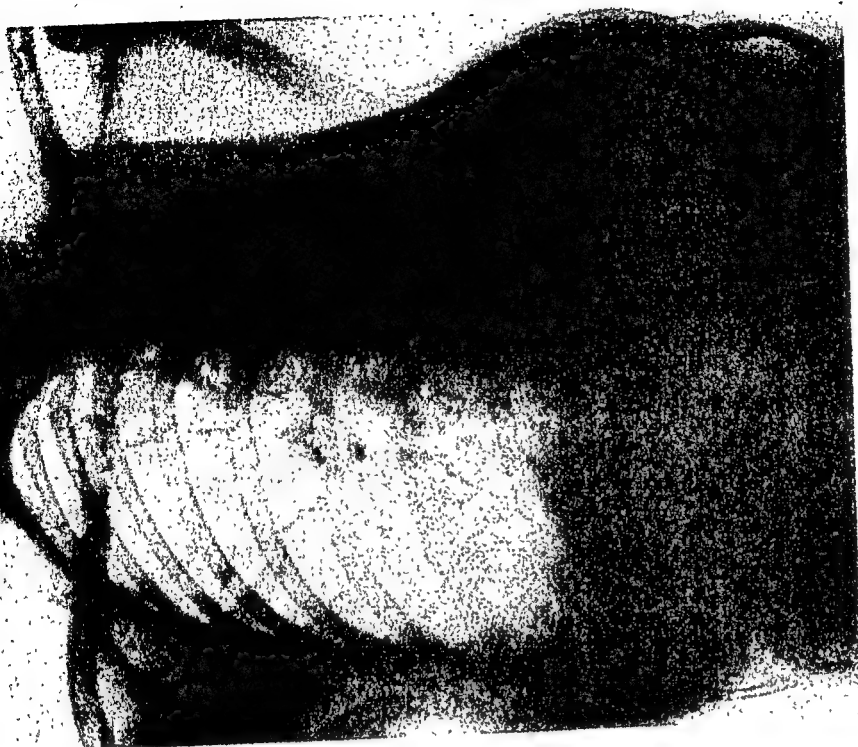


FIG. 327. Same case 21 months after complete left thoracoplasty. (clinical cure.)

The operation has completely cured many tuberculous patients who in the pre-thoracoplasty era would have gone steadily downhill. There are many modifications of the technique, the surgeon deciding from the location of the cavity how many ribs will require removal to ensure complete collapse of the cavity. As in pneumothorax, the surgeon's object is to obtain selective collapse and interfere as little as possible with healthy lung tissue. Accurate localisation

is most important, and this is much better achieved by tomography than by routine radiography. As there is considerable pleural thickening following a thoracoplasty, tomography is really the only method of revealing the effect of the operation on the cavity.

Immediately after the operation of thoracoplasty there is a fair amount of surgical emphysema, and on a plain radiogram a collection of air in the soft tissues may closely simulate a large uncollapsed cavity. I have seen such a collection of air in the soft tissues with a little



FIG. 329.—The right lower lobe was removed for bronchiectasis 12 months previously.

serum forming a fluid level, so superimposed on the mediastinal shadow as to simulate a mediastinal abscess.

Following thoracoplasty, the ribs tend to regenerate very quickly, thin strips of new bone forming in the course of a few months. This new bone is not very strong and fractures with slight trauma. These fractures are easily shown up and are treated like ordinary rib fractures. There is considerable pain at the time of the fracture, and this may lead to suspicion of a spontaneous pneumothorax.

LOBECTOMY AND PNEUMONECTOMY

Removal of a lobe or a lung may be carried out for bronchiectasis or carcinoma. The operative mortality is steadily decreasing, and the results in

bronchiectasis are most successful. Before operation for bronchiectasis a careful lipiodol examination of the whole of both lungs must be carried out. Only one lung is investigated at a time, so that a good lateral lipiodol picture may be obtained. The major bronchial arborisations in each lobe must be shown up clearly. The necessity for this precaution is obvious, as it is futile to remove a lobe or a lung if the other side is diseased. Following a lobectomy of the lower lobe, the chest wall shrinks a little and the upper lobe overdistends to fill the thorax. There is usually a little pleural thickening at the base and slight displacement of the heart and mediastinum to the affected side. As a result of the overdistension of the lung the finer vascular markings near the periphery are absent, and, of course, the total number of vessels seen is diminished.

The bronchial stump at the site of the resection dilates a little and, if subsequent lipiodol injection is made on this side, the lipiodol-filled stump may be mistaken by the uninitiated for a fresh bronchiectatic cavity.

Following a pneumonectomy, the chest wall falls in considerably, and when the wound heals and the air in the empty space is absorbed, the whole of the hemithorax appears opaque. This opacity is intensified by the thickening of the parietal pleura which usually follows a pneumonectomy. The heart and mediastinum are not grossly displaced following this operation.

PART THREE

URINARY AND MALE GENITAL TRACTS

BY

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PART THREE

URINARY AND MALE GENITAL TRACTS

CHAPTER XXXVII

THE URINARY TRACT: TECHNIQUE

GENERAL REMARKS

THE URINARY tract is examined in each of its regions by plain radiography or, more commonly, by a combination of plain radiography and contrast medium urography. The precise routine must be determined by circumstances. For the satisfactory radiographic investigation of these cases the co-operation of radiologist and urologist is most desirable. In some cases the services of the urologist are indispensable, as when cystoscopy and catheterisation of the ureters is required. But even in those in which such instrumentation is not adopted, the decision to be content with less elaborate methods of radiographic investigation can be properly arrived at only by consultation between clinician and radiologist.

The ideal arrangement is when the radiologist and urologist are both so placed as to be able to follow the investigation through to its completion at one sitting. This is not always possible, but its achievement is simplified if the patient be in an institution in which there is a combined X-ray and operating theatre for urological cases.

In such circumstances, inspection of the preliminary plain radiograms will indicate whether the use of contrast media is desirable. If so, they are applied forthwith *secundum artem*, and the further radiography immediately carried out. In the cases where the use of contrast media involves cystoscopy, the collection of pathological specimens, biopsy, etc., are dealt with at the same time.

In many cases, however, plain radiographic investigation only is carried out at the first sitting (this applies particularly in the case of suspected calculus); on the results of this examination it is decided whether further measures are needed. On the whole, this is a time-wasting method, since in few cases only does the additional use of contrast media fail to afford further essential information. Since the introduction of intravenous urography, the use of that method offers so little extra inconvenience as almost to warrant its use as a routine measure, in the absence of spec-

contraindications in the case. Indeed, such a routine is now the practice of many urologists.

In an increasing number of cases the routine adopted is first plain radiography, followed immediately by intravenous urography. Then, if the latter fail, as it sometimes does, to elucidate the condition of the renal pelves, instrumental urography is carried out on a subsequent date. This seems to be a reasonable compromise between the two systems of technique.

PREPARATION OF THE PATIENT

Preparation for Plain Radiography.—In many cases, where the available time is too short to allow of any preparation, the examination proves to be satisfactory without it. In an appreciable percentage, however, the examination is unsuccessful without preliminary preparation, because of obscuration of the film by faeces and gas. Gas in the intestines is the particular bugbear of genito-urinary radiography. With modern low kilovoltage technique, a large collection of gas in, for example, the splenic flexure may entirely blot out the left renal area. Faeces tend to obscure the pelvic view particularly.

There are various measures to be adopted to get rid of these obstacles to successful radiography:

Aperients constitute the most important item in the preparation, and are best taken on the evening before the examination. Those recommended are many, and on occasion almost any will prove satisfactory. If the patient is in the habit of taking an aperient, he should take rather a larger dose of the one to which he is accustomed. Cathartics should be avoided. They are distressing to the patient, and offer no advantage over gentler measures. They are also said to promote the formation of gas in the colon, but this is very problematic. It is difficult to see by what chemical process cathartics could have this effect. Favourite aperients in the preparation of these cases are castor oil, aloin, cascara, and phenolphthalein.

Enemata.—An enema is of use when an aperient has failed to have the desired effect. It should not be large, as if the whole colon is filled some of the enema may not be returned. The enema given should be a small soap-and-water one, to fill the rectum and sigmoid only, and by stimulation of the lower bowel to promote general colonic evacuation.

Colonic Lavage.—If aperients and small enemata fail to empty the colon satisfactorily, colonic lavage should be used. The Plombière method, which necessitates repeated visits to the w.c. and reinsertions of the rectal tube, is tedious, and a more efficient and less exhausting method is that of the Studa chair. By this method the colon is continuously irrigated, and the patient empties the bowel intermittently past the small metal rectal tube while sitting in the chair.

Posture is of considerable importance in the prevention of gaseous distension. Patients confined to bed show a marked tendency to collect gas in their intestines. Some of this may be due to the patient's condition, and so be unavoidable, but in a large percentage of cases the tympanites is due to air swallowed in the recumbent position. The patient is unable to get rid of the swallowed air by eructation, and that posture also facilitates the passage of swallowed air through the pylorus. Small quantities of air are usually swallowed along with saliva in between meals, and it is not enough that the patient should sit up in bed at mealtimes. Whenever it is possible, the patient should not be confined to bed during the twenty-four hours previous to the X-ray examination.

Taka-diastase—a carbohydrate ferment—is said to prevent digestive gas formation. Two to three tablets (gr. $2\frac{1}{2}$) may be given during each meal on the day before.

Pitressin, given hypodermically in doses of $\frac{1}{2}$ c.c. one and a half hours and half an hour before the examination, is often very effective in dispelling colonic gas. It should be used with caution, and larger doses, e.g. 1 c.c., are apt to cause colic and distress.

Diet.—This should be as free from residue as possible.

Drugs.—No drug containing an element of a high atomic weight should be taken for at least two days prior to the examination. The commonest sources of trouble in this respect are the bismuth and antacid powders so frequently given in dyspepsia.

A satisfactory preparation, then, should comprise an effective aperient on the day before, followed by an enema or colonic lavage on the next morning, if necessary. During the day before the examination the patient should be up and about if the clinical condition permits, and the diet should be one giving little residue.

Often satisfactory radiographic results are obtained in the absence of all these precautionary measures, but they should be applied if circumstances permit.

The bladder should *not* be emptied before the examination unless it is uncomfortably full. In the investigation of vesicle calculus it may be desirable to have the bladder well filled with fluid.

The preliminary preparation for instrumental pyelography is the same as that described above. The additional measures necessary for intravenous urography are indicated under that section.

ROUTINE TECHNIQUE

The minimum routine technique permissible in any case requiring urinary X-ray examination is that of two films, one of the renal and upper ureteric area, and one of the lower ureteric and vesical area. Any variation of

technique should be an elaboration thereof, such as stereoscopy, inspiratory and expiratory films, and radiograms taken after the use of contrast media.

The General Renal View should be taken with the patient supine on the Bucky grid, with a kilovoltage of not more than 70. If one picture only is taken, it should be exposed with the patient's breath held after expiration. The tube is centred midway between the umbilicus and the xiphisternum. Compression of the patient's abdomen with a wool pad is desirable, as it limits involuntary respiratory or other movements and reduces the thickness of the part through which the rays must travel.

If the patient is unable to understand what is meant by holding the breath—a not unusual experience—the instruction to close the lips and hold the nose is invariably successful.

In a radiogram of the renal areas the following structures should be shown, if the picture is to be regarded as of satisfactory technical quality.

(1) **THE OUTLINE OF BOTH KIDNEYS** in their entirety. These are, if anything, better seen in fat subjects, as the perirenal fat acts as a contrast medium. Gas in the colon, and, to a lesser extent, in the small intestine may prevent the outline being visible, in spite of satisfactory technique.

(2) **THE USUAL BONY STRUCTURES** should be clearly visible, including the last two ribs above and the iliac crests below. The delicate structure of the transverse processes of the lumbar vertebrae supply a good test of the quality of a radiogram.

(3) **ANY GAS IN THE STOMACH**, small intestine, and colon should be sharp in outline. Blurring of this usually indicates respiratory movement on the part of the patient.

(4) **THE OUTER BORDER OF THE PSOAS MUSCLES**

The Vesical View is taken in a similar manner, except for the centring point. The tube may either be centred perpendicularly over a point midway between the anterior superior iliac spines, or may centre on that point with 5–10 degrees of caudal angulation. The latter view throws the shadow of the symphysis clear of the prostatic area. It is important that this view should be a true antero-posterior one, particularly when there is a doubtful shadow in the ureteric line. Slight lateral rotation causes considerable distortion, and renders it difficult to plot out the normal ureteric line even approximately.

The vesical view should include the iliac crests and the entire true pelvis, and should cover an area well below the lowest point of the symphysis pubis. A satisfactory radiogram of the vesical area should show a sharp outline to any gas in the sigmoid and rectum, and in the majority of cases the bladder outline is visible.

A lateral view of the renal areas is a feasible and useful adjunct to the antero-posterior, especially in the differentiation between renal and biliary calculi. A true lateral view of the pelvis is of no value, because of the superimposition of both hip joint regions. As a compromise, right and left semilateral views

are often helpful. They find their particular use in vesical diverticula and in urethrography.

The presence of a doubtful shadow in the above preliminary examination will at once call for confirmatory examination. These confirmatory methods include :

The Lateral View of the renal area. This is of value in determining the coronal plane of shadows. Those of renal origin are superimposed on the shadow of the lumbar spine in this view.

Stereoscopy of the Pelvic View.—Stereo-radiograms are of great value in determining the relative position of pelvic opacities such as calculi, phleboliths, or calcified glands. The pelvic basin affords admirable landmarks against which to localise these shadows.

Radiograms taken on Expiration and Inspiration.—The object of these is to establish the respiratory range of movement of an opaque body seen in the renal view. As other bodies beside calculi show a similar respiratory travel, the test is little used now. Much more reliable methods are now available.

CHAPTER XXXVIII

UROGRAPHY

THE TERM "urography" is applied to the use of contrast media for the radiographic demonstration of the lumina of the urinary tract and adjacent male sexual organs. The relationships of adjacent sinuses and fistula may be demonstrated either by normal urographic methods or by injection through the fistulous openings.

Two main methods have to be considered—instrumental and intravenous.

INSTRUMENTAL UROGRAPHY

The term includes a variety of investigations, depending on the particular part examined, as follows: pyelography, ureterography, cystography, urethrography, vesiculography. Much the most important and most frequently used are the first two, usually included in the term "pyelography"

PYELOGRAPHY

Instrumental, ascending, retrograde, or transvesical are the adjectives used to distinguish this from the intravenous method

The Media.—*Klose* is credited with the first attempt at pyelography. He used a suspension of bismuth.

Welcher and Lichtenberg were the first to use the silver preparations successfully. They employed 2 per cent. collargol. Following on this, other silver compounds were tried, argyrol and silver iodide. The objections to the silver media were several. A number of fatalities and lesser accidents occurred from renal necrosis, silver poisoning, and silver embolism. These, and a tendency to staining of towels, etc., led to their being supplanted by thorium nitrate, in 10 per cent. neutral solution. This substance is irritant to the mucous linings, particularly to the bladder.

In 1918 the halogen compounds were introduced, first sodium bromide, and later sodium iodide. The latter was soon generally adopted. The points in its favour are that in a concentration of 15 per cent. it is non-toxic, adequately radio-opaque, easily sterilisable, and low in viscosity and osmotic pressure. The more hypertonic solutions of, say, 30 per cent., cause irritation and œdema of the delicate mucosæ. The solution may be sterilised either by boiling or by the addition of mercuric iodide to make a 1 in 3,000 solution.

Lithium iodide has been tried and given up on account of its irritant qualities.

Uroselectan B. makes an excellent medium for instrumental pyelography where the utmost detail is required, as in the diagnosis of a renal neoplasm, or the demonstration of tuberculous erosion of a calyx. Admirable results may be obtained by the use of a 20 per cent. solution, sterilised by the addition of mercuric oxycyanide to a strength of 1 in 100,000. The only objections to the use of Uroselectan B. are its cost and its viscosity. Recently a weak solution, one-fifth of the strength of the intravenous preparation, has been prepared in sterile ampoules. ready for instrumental use.

Thorium dioxide has also been used recently as a contrast medium with satisfactory results. "Thorotrast," a 25 per cent. suspension of thorium dioxide, is a satisfactory preparation. It is supplied in 12-c.c. ampoules for instrumental pyelography, and in 100-c.c. containers for cystographic use. It is claimed that it is less irritating than the iodine preparations. Its density is a desirable property, but its viscosity—greater than that of any of the halogen preparations—makes it difficult in some cases to inject.

Another satisfactory medium is a solution of sodium ortho-iodo-hippuric acid, now available under the name "Iodoray" in 10-c.c. ampoules, each containing 3 grammes of the drug.

TECHNIQUE OF INSTRUMENTAL PYELOGRAPHY

Anæsthesia.—General anæsthesia is contraindicated by two considerations. It is desirable that the patient be conscious, as a guard against renal damage by overdistension. Pain in the loin gives an immediate warning of this. Again, the patient must hold his breath during the exposures if satisfactory radio-grams are to be taken. The makeshift of forcibly closing the mouth and nose of the anæsthetised patient rarely succeeds in immobilising the kidney sufficiently. A local urethral (or low spinal) anæsthetic is not required in the female, but is usually necessary for the cystoscopy in the male. Novocaine, or one of its derivatives, such as kerocaine or percaïne, gives satisfactory anæsthesia.

The Injection of the Medium.—The solution chosen is injected at body temperature. The amount to be injected may be determined by the following considerations:

(a) **THE CAPACITY OF THE RENAL PELVIS**, which is stated to range, in the normal, between 6 and 14 c.c. This is far from accurate; it may be much more—two or three times as much. The figure given above expresses the quantity which may be injected slowly in the conscious patient without inducing pain in the loin.

(b) **THIS PAIN** affords a valuable index of the amount which may safely be injected, but in hydronephrotic cases the pain is not reliable, and if such a condition is suspected, a preliminary estimation of the renal capacity may be attempted. With the catheter in situ in the renal pelvis, the contents of

latter are aspirated. The renal pelvis is then filled with saline, gravity pressure being used, and the saline then withdrawn and measured.

Preliminary emptying of the renal pelvis is desirable from a radiographic standpoint. It prevents dilution of the contrast medium and adds sharpness to the pyelographic contour. It may, however, increase the irritation of the injection. The attempt at aspiration also demonstrates whether the end of the catheter is impacted into a pyramid.

The ideal method of injection is with the cystoscope still in situ. The renal pelvis is filled. The catheter is then withdrawn to the lower end of the ureter and the latter filled by a further injection of a few cubic centimetres. The X-ray exposures are then made. As the contrast medium frequently leaks past the catheter into the bladder, it may be necessary to keep up gentle pressure on the syringe during the exposure, to ensure proper visualisation of the ureters.

The X-ray investigations being completed, the ureteric catheters are reintroduced an adequate distance into the ureters, the cystoscope removed, and the urine collected for pathological examination. If inspection of the first pyelogram indicates that more are necessary, the retention of the catheters enables a further injection to be made for this purpose.

If the examination has been prolonged, it is probable that the bladder is full of a relatively opaque mixture of urine and contrast medium. This may obscure the lower end of the ureter, and if a late ureteric radiogram of this portion is required, the bladder should first be emptied.

INTRAVENOUS UROGRAPHY

This method, consisting of intravenous injection of a contrast medium of such nature that it is excreted in the urine in sufficient amount to cast a shadow of the renal pelvis, ureters, and bladder, was first introduced as a practical routine method by *von Lichtenberg*.

At first hailed as almost a complete supplanter of the instrumental method, it now occupies a less exclusive though still valuable position in the examination of the urinary tract. As a method it has both advantages and disadvantages; these may be relative, or in some cases absolute.

Indications for the Use of the Method.—There are certain *absolute indications*, in cases where the instrumental method is impossible, impracticable, or dangerous. Such cases include urethral stricture, cystitis, vesical hæmorrhage, colonic transplantation of the ureters, and those in which for any reason cystoscopy is refused. In this group intravenous urography forms a welcome alternative. It is particularly valuable in children.

Apart from absolute indications, the method offers certain *relative advantages*, such as where it is desired to demonstrate the ureters undistorted by the presence of a ureteric catheter, or, *per contra*, the tortuous ureter unstraightened by the catheter. Again, the method affords some indication of renal function. In this respect its precise value has still to be assessed; the blood urea is, so

far, a surer guide. In advanced renal failure the method gives no qualitative indication, as no visible excretion occurs at all. Other advantages of the method are its relative painlessness, greater ease of application, and that it is available in those cases where double ureteric catheterisation is considered inadvisable.

Contraindications.—The following are generally accepted as *positive contraindications* to the method: severe renal and hepatic insufficiency, acute inflammations of the kidney, advanced cardiac disease, thyrotoxicosis, and severe anæmia. If used in the presence of these conditions, a preliminary estimation of the blood urea is desirable, as the possibility of precipitating an uræmic crisis by the injection is very likely. (This observation does not, of course, apply to the cases where *one* kidney is destroyed and the other healthy; but here the invisibility of the diseased organ vitiates the usefulness of the method.) Pregnancy does not constitute a contraindication, except when complicated by one of the preceding.

There are several *disadvantages* associated with the method. The most serious of these is the poor contrast compared with an instrumental pyelogram. In cases where the accurate demonstration of a small pelvic or calyceal lesion is necessary, intravenous pyelography may be valueless. In addition to the shadow frequently being faint, the filling of the calyceal and pelvic lumina is often incomplete. Again, in many gross renal lesions no shadow at all appears, while demonstration of the ureters in their entirety is often not achieved. The best shadows are obtained in cases of urinary obstruction without gross impairment of functions.

The Medium.—Intravenous pyelography was first attempted by *Rowntree*, using sodium iodide. The concentration in the urine proved far too low to give practical results. Later *Binz* and *von Lichtenberg* investigated a series of iodine-pyridin compounds, including Selectan, Skiodan, and Noiopax. Success was first achieved with Uroselectan, Abrodil, and Per-Abrodil.

Finally, Uroselectan B. was introduced, and this and Per-Abrodil are now used almost universally.

Uroselectan B., a product of the German firm of Schering-Kahlbaum, was introduced in 1930 as an improvement on their original Uroselectan product. The advantages it possesses over the former are lessened toxicity, smaller bulk, and greater radiographic contrast.

CHEMISTRY.—The makers give the formula of Uroselectan B. as the disodium salt of 3·5-diiodo-4-pyridoxyl-N-methyl-2·6-dicarboxylic acid. Its molecular weight is 493, and it contains about 51 per cent. of iodine.

The standard dose is 15 grammes dissolved in 10 per cent. glucose solution to a total bulk of 20 c.c. It is sold in sterile ampoules, ready for intravenous injection.

The drug can be demonstrated in the urine by adding one part of concentrated HCl to four of urine. Crystals of dicarboxylic acid are precipitated with liberation of CO₂. The crystals have a melting-point of 174° C.

PHARMACOLOGY.—*Gardner and Heathcote* have made a detailed study of this, and investigated its effect in rabbits and dogs.

The recommended intravenous dose in human beings is 0.25 gramme per kilogramme body weight. This, when injected, gives a concentration of 0.3 per cent. in the blood.

They found that the isolated heart and intestines of the rabbit are unaffected when perfused by a solution of the above concentration.

In anæsthetised dogs, injections of the drug up to 1.25 grammes per kilogramme produced only mild changes in the blood-pressure—a slight drop, followed by a prompt return to normal. The respiratory rate and depth were increased by these doses. Larger doses had an increasingly depressant effect, but the drug is relatively non-toxic to dogs, which, it was found, could survive an intravenous dose of 5 grammes per kilogramme of body weight.

Repeated intraperitoneal injection of 1.25 grammes per kilogramme was found to produce fatty degeneration of the liver and cloudy swelling of the kidney.

In their experimental urography in dogs, *Gardner and Heathcote* found that the Uroselectan B. shadow was about the same density as that obtained with Abrodil, but that it appeared much sooner. This latter observation is in accord with clinical experience in man: the shadow is, however, generally held to be denser than that given by the earlier drugs.

EXCRETION.—Uroselectan B. is excreted unchanged in the urine. According to *von Lichtenberg* 30 per cent. of the drug is excreted during the first hour, in the second 12 per cent., and in the next six 21 per cent. A diuresis is produced during the first fifteen minutes. This rapidly dies down, so that the maximum concentration of the drug in the urine occurs during the second period of fifteen minutes. The maximum radiographic contrast is obtained during the period ten to thirty minutes after the injection.

Von Lichtenberg gives the maximum concentration found in the urine in man as 7 per cent., and in the average case 4–5 per cent. represents the peak concentration.

Gardner and Heathcote found that the excretion of Uroselectan B. is greatly reduced and retarded in uranium nephritis. This, likewise, agrees with clinical evidence.

Per-Abrodil.—Another drug which gives satisfactory results is that produced by the firm of Bayer under the name of Per-Abrodil. The formula of this is given as 3:5-diiodo-4-pyridone-N-acetate of diethanolamine. It is stated to contain 51 per cent. by weight of iodine. It is supplied in sterile solution in 20-c.c. ampoules containing 7 grammes of the drug, ready for intravenous injection. The dose for an adult is 20 c.c., for children 8–10 c.c., and for infants 2–3 c.c.

The rate of excretion and radiographic properties are very similar to those of Uroselectan B. Thus, in the normal subject, the optimum shadow is

obtained eight to twenty minutes after injection. The makers give as contra-indications to the use of the drug severe renal impairment and severe systemic disease. Renal tuberculosis and pregnancy do not preclude its use.

Sodium Ortho-iodo-hippurate.—In 1933 *Moses Schwick* introduced this drug for intravenous urography[†] (and also for oral urography). It is a derivative of a salt normally found in the urine, and, as was anticipated by *Schwick*, has proved to be non-toxic, highly soluble, neutral in solution, and to be rapidly excreted by the kidneys. Its iodine content is 38.8 per cent., considerably lower than that of Uroselectan B. or Per-Abrodil.

Schwick recommends a dose for adults of 10–15 grammes dissolved in 20–30 c.c. distilled water. Maximum concentration in the urine occurs at ten to twenty minutes after injection—rather more slowly than the other two drugs mentioned. Children tolerate the drug well.

The drug has been prepared in convenient form in this country by *Martindale*, under the proprietary name of “Iodoray.”

TECHNIQUE FOR INTRAVENOUS UROGRAPHY

Preliminary Preparation of the Patient.—The patient should undergo the routine preparation described above for any renal examination, and in addition should have his fluid intake restricted for at least twelve hours. The object of this last is to make the urine as concentrated as possible at the time of the examination, and so increase the density of the shadows obtained. The production of diuresis prior to the examination by the exhibition of urea has been recommended, but if this be used the urea must be given not less than six hours prior to the injection, as the diuresis produced thereby may last for that time.

The injection should be made slowly into a suitable vein such as the median basilic or median cephalic. It is important that the injection be slow. As long as two to three minutes may with advantage be occupied by the injection, with the object of avoiding systemic reaction. Although of course undesirable, no such disastrous results follow leakage of the injection into the subcutaneous tissue, as is the case with tetraiodophenolphthalein in intravenous cholecystography. Paravenous extravasation of Uroselectan B. is followed by pain and some local swelling for a few days. With Per-Abrodil there is no reaction at all, and according to *McAlpine* this drug may be given subcutaneously in children if a suitable vein cannot be found.

The fluid should be warmed to blood heat before administration.

Immediate Sequelæ.—The patient frequently experiences transitory symptoms during and immediately after the injection, such as flushing of the face, elevation of the pulse-rate, general feeling of warmth, pain or disc- in the arm and shoulder on the side of the injection, thirst and dryness the throat, and occasionally giddiness, faintness, and singing in the ears. These, as a rule, rapidly pass off, unaided, or with the help of a small

sal volatile The pain radiating up the arm and shoulder is said to be due to venospasm induced by the drug.

Radiographic Technique.—Several points should be borne in mind in considering the radiographic technique. The available contrast is not great ; consequently the technique must be directed to making the most of it, such as it is, by use of the Bucky grid and a low kilovoltage. Speed of exposure must not be sacrificed too much in achieving this.

As the method is a test of function as well as of morphology, a series of radiograms should be taken to demonstrate the various phases of secretion, and as the secretion of the drug rises to a peak during the first fifteen or twenty minutes, several of this series should be taken during that interval. A useful routine series is that taken at the following times after the injection : 3, 7, 15, 20, 30, 60 minutes. Strict adherence to times is not necessary.

In certain cases of impaired renal efficiency, notably hydronephrosis, secretion is considerably delayed, and it may be necessary to wait as long as six hours to obtain the maximum contrast. Any variation in the routine will be indicated by the appearance in the first two or three radiograms.

If it is particularly desired to study the morphology of the renal pelves, an attempt may be made to dam back the drug-laden urine in them by pressure in the hypogastrum. The pressure must be maintained for at least five minutes prior to the exposure. This can only rarely be successful, because of the severe pressure necessary to occlude the ureters.

As the ureters are demonstrated by this method, the radiograms should include them in their entire length. This applies to the radiograms taken at each stage, since only a portion of the ureter may be visible at a time.

In the later stages the bladder may obscure the lower ends of the ureter, and if these have not already been visualised, it is wise as a routine to take a final picture after emptying the bladder, to unmask the ends of the ureter and to determine the amount of residual urine in cases of enlarged prostate.

Often the ureteric shadows can be made out through the vesical, if the latter is not too dense.

Finally, the method usually affords a satisfactory cystogram.

THE NORMAL APPEARANCES IN INTRAVENOUS UROGRAPHY

After the injection of Uroselectan B the first sign in a radiogram of its excretion is the appearance of faint triangular outlines of the minor calyces. This may occur even before the injection is completed. Very shortly after the appearance of these, the renal pelvis becomes outlined, and then the connecting necks between pelvis and calyces. Gradually the pyelogram, in a successful case, fills out and its contours become defined, and at the same time the ureters become visible in parts. The portion of the ureter visible in any particular radiogram depends on the precise position of the peristaltic wave

driving the urine down into the bladder. The ureter is not normally filled along its whole length by intravenous urography; such a state of affairs, if found, should raise the suspicion of ureterospasm or ureteral obstruction. The upper portions of the ureters are more commonly seen than the lower. The mechanism of transmission of urine from kidney to bladder is described in detail later, but a study of serial Uroselectan B. radiograms shows it to consist in a gradual preliminary filling of the renal pelvis. When this becomes sufficiently distended, peristalsis is set up, partially emptying the renal pelvis, filling, and then more or less completely emptying the upper ureter. This upper

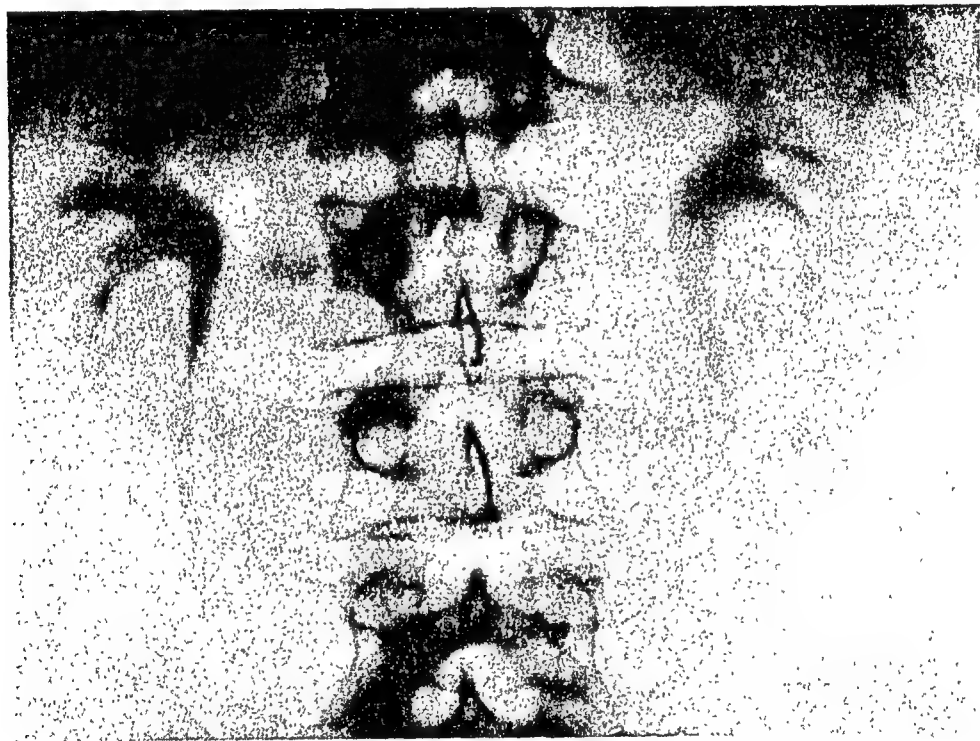


FIG. 329.—Normal Uroselectan B. pyelograms, 10 minutes after injection.

portion of the ureter corresponds to the upper ureteric spindle. The urine impelled down from this may remain for a short period in that dilatation of the ureter extending from the brim of the bony pelvis to the bladder. This is the so-called "middle" spindle. A third spindle is described as occurring in the last inch of the free ureter, and can occasionally be made out in a Uroselectan B. radiogram. This last inch may be obscured by the bladder containing opaque urine.

The density of the vesical shadow will depend not only on the concentration of the urine therein, but also on the degree of dilution thereof by urine previously contained in the viscus. The bladder should therefore be empty

prior to the injection if a cystogram be desired. As the bladder fills, it will be seen first in the form of an inverted pyramid, the upward-facing base tending to be concave. With increasing distension the superior surface becomes flat, then convex, and finally the bladder assumes a spherical or ovoid shape. The appearances are then identical with those of an instrumental cystogram, save that the density of the contrast medium is less. Frequently the vesical shadow is seen to be asymmetrical, particularly in the earlier stages of filling, an appearance which has as a rule no significance.

If transvesical urography is not possible because of a local condition, and it is impracticable to administer a contrast medium intravenously, e.g. in very young children, it is possible to give the contrast drug by two other routes, subcutaneously or by the mouth.

SUBCUTANEOUS UROGRAPHY

This was first attempted by *Butzengeiger* in 1931, who injected 500 c.c. of a 4 per cent. isotonic solution of Skodan, containing 20 grammes of the drug, into the axillæ. He reported thirty cases giving results almost as satisfactory as those obtained by the intravenous method. No harmful effects resulted. The maximum excretion occurred thirty to fifty minutes after injection. The objection is the size of the subcutaneous injection. In 1932 *Hillebrand* successfully used the method in a child, giving 100 c.c. of a similar solution.

An improvement on the method is that reported by *Baer* and *Theodore*, who used Neoskiodan in 7 per cent. isotonic solution. They recommend the injection of 50 c.c. into each axilla, giving a total dose of 7 grammes. Because of the low toxicity of the drug, the full dose may be given to infants. Local anaesthesia is advisable in older subjects, to allay the slight discomfort which follows the injection. The maximum concentration in the renal pelvis occurs fifty to sixty minutes after administration.

ORAL UROGRAPHY

The new contrast medium, sodium orthoiodo-hippurate, introduced by *Schwick* and *Jaches*, is suitable also for oral urography. These workers recommend a dose of 10–15 grammes dissolved in 75 c.c. of a syrup and lactopeptone mixture. They have found no unfavourable reactions to the drug. The maximum concentration of the drug in the urine occurs sixty to 120 minutes after ingestion. The density of the urographic shadow is considerably less than in intravenous urography, and is much less certain in its effect. A satisfactory cystogram is obtained in all cases, since the lessened density is balanced by the greater bulk in the bladder. A disadvantage of the method is the taste of the drug, which to some is unpleasant.

These authors recommend, in addition to the preparation usual for intravenous urography, the withholding of drugs such as potassium citrate and acid sodium phosphate for twenty-four hours prior to the examination.

CYSTOGRAPHY

The bladder may be filled through either a cystoscope or a catheter. The latter, being much the simpler process, is to be preferred, since its efficiency is little less than the other method. In cases where cystoscopy is a necessary diagnostic step, the cystography should be carried out at the same sitting.

The bladder should first be completely emptied, and then filled with an 8-10 per cent. solution of sodium iodide. It should be run in by gravity under a pressure of 6-12 inches of water. Too dense a solution is not desirable. If the medium be very opaque, half shadows of filling-defects and diverticula may be blotted out by the main vesical shadow, when a more transparent medium would allow them to be visible through the vesical opacity. Too poor a contrast is, on the other hand, to be avoided, lest a marginal irregularity be not clearly defined.

Antero-posterior and right and left oblique radiograms should be taken, in order to view as much of the bladder wall in profile as possible. These radiograms should be taken with the catheter or cystoscope in situ, not only because further radiograms may be necessary and a further injection required, but also because the instrument accurately marks in a radiogram the neck of the bladder.

Air has been used as a medium for cystography. For an air cystogram to be successful the bladder must have been completely emptied, and the rectum and sigmoid must be quite clear from faeces and gas, particularly the latter. Given these conditions, an air cystogram gives an excellent outline, but because of the difficulty of completely emptying the colon of gas, and more particularly the danger of air embolus, other contrast media are to be preferred.

Uroselectan B. is an admirable medium, if suitably diluted. It finds its chief place, however, in intravenous cystography.

Lipiodol Cystography.—A method has been described in which saline and lipiodol are introduced into the bladder. With the patient in a high Trendelenberg position the lipiodol floats against the now uppermost base of the bladder. The object of the method is to demonstrate basal filling-defects. The difficulties of maintaining the position and obtaining radiograms free from gross distortions preclude its general use.

VESICULOGRAPHY

This may be defined as the visualisation of the lumen of the seminal vesicle, vas deferens, and ejaculatory duct by the injection of a contrast medium. The objects of the procedure are to demonstrate the patency of those ducts and a distension or destruction of the seminal vesicle. Lipiodol is the medium most commonly employed. The disadvantage is its viscosity. This renders injection along the fine ducts a matter of considerable difficulty. Uroselectan

B. is a medium free from that objection. It gives radiograms of adequate contrast, and clinically it is without harmful effects.

Technique.—The only satisfactory method of injection is directly into the vas deferens, exposed by a small inguinal incision under local analgesia.¹ Urethroscopic catheterisation of the ejaculatory ducts, even in the hands of the expert, is very rarely feasible in the normal subject, and less so in disease. In cases particularly calling for its employment, it is rendered especially difficult by the pathological condition present (e.g. verumontanitis). In addition, injection is then dangerous on account of the risk of inducing epididymitis. Finally, urethroscopic catheterisation, even when successful, serves only to outline the ejaculatory ducts and the seminal vesicles. It is not possible to fill the vas deferens by this method. To effect this, direct injection into the vas deferens must be made, in both directions; upwards to fill the vesicles, and downwards to the epididymis, where the patency of the tubules may thus be investigated.

EXTRAVASATIONS

The radiographic demonstration of urinary extravasations such as may occur in rupture of the urethra, bladder, or kidney, has been advocated. The principle and technique are obvious; the opportunities rarely arise, and still less do the indications therefor.

The most satisfactory medium would be Uroselectan B.

ARTIFICIAL PERIRENAL EMPHYSEMA

This method was devised by *Caselli* and *Sordelli*, and *Rosenstein*, in 1921. It consists of the introduction of gas—oxygen or carbon dioxide—with the object of bringing the renal and adrenal outlines into sharper relief in a radiogram. As it is now possible to show the entire renal outlines in a plain radiogram in the vast majority of cases, the method, one not without risk, has fallen into disuse. It might, however, be of use in demonstrating the contours of the adrenals—e.g. in cases of adrenal virilism. It is therefore described here.

Quinby has given the following account of the technique.

"Oxygen is collected in a sterile litre flask connected by rubber tubing both with another flask twice this size, containing sterile water, and with an ordinary lumbar puncture needle marked in centimetres. With the patient lying on the side, after local anaesthesia, the needle is introduced into the loin,

¹The vas deferens is firmly held subcutaneously by the left index finger and thumb, the novocaine injection and the small incision being made without relaxing this grip—the vas is then hooked out and kept out of the incision by a thread or ligature. This is much easier and more devoid of complication than dissection of the spermatic cord. The tissues down to the vas are separated after the incision by opening the blades of a forceps. The right hand uses in turn—anaesthetic syringe, scalpel, forceps, aneurysm needle, fine tenotome to make small longitudinal incision in vas, syringe for injecting.

pointing upward and inward toward the lower pole of the kidney. The distance to which the needle must penetrate varies greatly in different individuals, the extremes being from 4 to 10 cm. The tip should go just outside the erector spinæ group of muscles, traversing the intrinsic muscles of the abdominal wall very near their origin. In order that the injection may be successful the needle tip must always pass through the fascia renalis into the true perirenal fat. If it remains outside this fascia in the retroperitoneal fat layer the kidney will not be outlined. Furthermore the needle tip must not penetrate the kidney itself. It is in regard to this precise position which must be obtained that most of the negative results have been due, because the amount of perirenal fat tissue varies greatly in different individuals and in different conditions of the kidney.

"The needle being placed in position, the larger of the two flasks is inverted and elevated, and thus the water flowing into the oxygen-filled flask displaces the gas outward through the needle. When the needle is properly placed the inflow of gas is obtained with only moderate pressure. If the flask containing water has to be elevated more than a couple of feet above the level of the gas containing flask the needle is not properly placed and should be immediately readjusted. Injection is stopped after about 500 c.c. of oxygen have been displaced by water.

"If oxygen is the gas which is used, the best results are obtained by making the röntgen-ray exposure about twelve or fourteen hours after the injection of the kidney pelvis. This is due to the fact that oxygen is absorbed from these tissues with relative slowness, and by waiting this length of time the gas is given opportunity to diffuse evenly throughout the perirenal area. We frequently found that in those röntgen-ray films made immediately after injection the gas was so densely located about the region of the lower pole of the organ that the kidney shadow here was either much deformed or even quite invisible. This condition may not occur if carbon dioxide is used because of its greater diffusibility and quicker absorption, but I have not cared to use this gas in such quantities as are here necessary, thinking that it might be more dangerous."

The risk of the method would appear to be that of penetration of the gut or large blood-vessels (in competent hands this should be nil), and the chief difficulty to estimate the distance to which the needle must penetrate to reach the perirenal plane. It is noteworthy that the last publication of importance on this subject was that of *Quinby* in 1923.

CHAPTER XXXIX

THE KIDNEYS

NORMAL X-RAY APPEARANCES

IN NO organ of the body is there such great variation within the compass of the normal as in the case of the kidney. The utmost care must therefore be exercised in deciding whether a given variation falls within normal limits or is pathological. Such variations occur in connection with

- (1) The outline of the kidney
- (2) The size.
- (3) The position and mobility.
- (4) The form of the calyces
- (5) The form of the renal pelvis
- (6) The origin of the ureter.

The Outline of the Kidney is visible in a radiogram because of the relative translucency of the perirenal fat. The latter forms a contrast medium, and the outline is often particularly well seen in obese subjects. The renal outline should always be clearly visible, and a radiogram which does not show it should be regarded as being below the necessary diagnostic quality. The normal shape or outline approaches to the conventional reniform shape, but not accurately. The upper pole is often rather pointed compared with the lower, and the breadth of the renal shadow is often lessened by the foreshortened view resulting from the obliquity of the renal plane to the coronal plane of the body. The outer border should be regularly convex, sweeping without any irregularity into the poles and on into the inner border. The outlines disappear as the hilum is approached. Irregularities in or disappearance of the outline may have a diagnostic value in the following conditions: tumour or cyst, horseshoe deformity, polycystic disease.

The Size of the Kidney should be noted. There is often a moderate difference in size of the kidneys in the normal subject. A considerable enlargement may result from compensatory hypertrophy if the other be destroyed. Again, considerable enlargement may be the result of tumour or cyst formation, or hydronephrosis. Gross enlargement may be due to polycystic disease (in which case it is bilateral) or hydronephrosis. In children, sarcoma is a likely cause.

The Position and Mobility of the Kidney, as stated in anatomical textbooks, is one in which the pelvis is opposite the transverse process of the first lumbar vertebra on the right side, and on the left a little higher. In the light of radiographic demonstration this position is shown to be too high. An average level

for the right kidney is with its hilum opposite the second lumbar vertebra, and the left kidney $\frac{1}{2}$ to 1 inch higher. The radiographic position will vary according to whether the exposure is made in full expiration or inspiration. If the kidney shadow be much below this level, the conditions of ectopia and ptosis call for consideration. The long axes of the kidneys are obliquely disposed to each other, the upper poles being a trifle nearer together than the lower.

The Pelvis and Calyces.—It is here that the kidney shows its greatest variations. In this respect the organ is veritably protean. The multiplicity of appearances that may be seen in the normal pyelogram constitutes a serious difficulty in the diagnosis of pathological lesions, a difficulty rendered more acute by the fact that the two kidneys in any one subject may show wide individual variations.

There are recognised in the renal pelvis two extreme types, with all degrees of transitional forms. In one there is a well-formed renal basin, and in the other the ureter divides without expansion into minor calyces. In the great majority of cases, however, an ampulla or pelvis is present (Figs. 330, 331, and 332).

THE CLASSICAL RENAL PELVIS is trumpet-shaped, the trumpet having an outward bend. It merges below with the ureter, no point of demarcation being evident. The outer margin of the ureter and the lower margin of the renal pelvis form a gentle regular concavity, without any angulation. Similarly the inner ureteric margin and upper pelvic margin form a regular convexity. These appearances are present only if the organ is viewed in profile. Above, in the classical type, the pelvis divides into three primary or major calyces—cephalic, middle, and caudal. The two latter are frequently close together and may form one major calyx. Each of these finally subdivides into an anterior and posterior row of minor calyces. The number of minor calyces is very variable. On an average there are four springing from the cephalic primary calyx, and six from the middle and caudal. The major calyces are merely channels. The minor calyces consist of a neck and an expanded extremity, cupped by the projection of the renal pyramid into its lumen. It is obvious that the outline of the minor calyces in a pyelogram will depend on whether it is seen in profile, *en face*, or intermediately. As "clubbing" of the calyces is an important sign of hydronephrosis, it is important not to mistake the rounded shadow of a calyx seen end on for a clubbed one.

BIFID PELVIS. DOUBLE URETER.—The division of the renal pelvis into two primary calyces may be exaggerated, up to a complete division of the forme down to the ureteric insertion. The appearance is then known as a bifid pelvis. The division may extend down the ureter, and produce a partial or complete double ureter.

IN A THIRD TYPE the renal pelvis fails to divide into primary calyces. It is then larger, and the minor calyces bud off its outer border.

THE SITUATION OF THE RENAL PELVIS relative to the kidney substance, be determined when the renal outline is visible in a pyelogram. Again

siderable variation may occur between extremes in which the pelvis is entirely extrarenal and entirely intrarenal.

In cases in which the kidney lies obliquely against the spinal column and so presents a three-quarter view in the standard antero-posterior radiogram, the



FIG 330 — Normal ascending pyelo ureterogram, showing the ureteral spindles. A gas bubble is present in the lower calyx.



FIG 331 — Normal ascending pyelo ureterogram.

pyelographic appearance is considerably altered. The ureteric shadow ends abruptly and eccentrically in an oval blob—the renal pelvis, from the outer border of which spring the calyces. Some of the latter are seen end on and appear as rounded shadows, perhaps with a translucent centre, where the pyramidal apex projects into them. Others may be hidden by the pelvic shadow.

The Ducts of Bellini.—When the injection of the opaque medium into the renal pelvis has been vigorous—frequently indeed when no undue force has been used—the injection—a certain amount may pass into these ducts, and be visible as a radiating feathery spray from the centre of each minor calyx.



FIG. 332.—Normal ascending pyelogram.



FIG. 333.—Normal ascending pyelogram showing the "snow-plume" effect when the medium (Uroselectan B. in this case) passes into the collecting ducts of Bellini. Some of the medium appears also to have escaped into the arcuate veins.

This happens more readily when Uroselectan B. is used as a medium for instrumental pyelography. It has no pathological significance (Fig. 333).

PELVIC RESORPTION

In a certain number of cases of pyelography, when the renal pelvis has been well filled or distended, the opaque medium escapes from the

There appear to be two routes by which it does this, via the lymphatics and into the renal veins, the latter being termed by *Hinman* and *Lee Brown* 'pyelovenous backflow.'

The Renal Lymphatics.—When the drug is absorbed by the lymphatics they become visible as irregular lines rising from the region of the hilum inwards to the renal glands and upwards and inwards to the celiac group. The writers have not observed this phenomenon with sodium iodide injections in normal subjects.

Abeshouse holds the view that lymphatic backflow occurs as the result of easy penetration of the injected fluid into abnormally patent renal lymphatics, either congenital or acquired. The acquired type is said to occur from obstruction of the thoracic duct, with resulting dilatation, tortuosity and finally rupture of the renal lymphatics. It is associated with chyluria. The congenital type is postulated by *Abeshouse* to cover those cases in which chyluria was not present, but proof of the existence of these congenital patent lymphatics is not yet available. *Higham*, who has recently reviewed the subject, is of opinion that in all cases in which lymphatics are visible in a radiogram, the medium has been absorbed from the perivascular lymphatic spaces. A full bibliography is given in his paper.

Pyelovenous Backflow.—There seems to be no doubt that in some cases the injected medium can escape into the renal veins. The radiographic picture is one of a faint hazy streaky shadow surrounding the major calyces and the necks of the minor. The appearance is as though thick cobwebs had accumulated around a pyelographic cast,

obscuring to some extent the intercalycal space (Fig. 334). *Hinman* and *Lee Brown*, who have investigated this matter fully, note that in making celloidin-corrosion preparations of the kidney some of the venous system was also filled, and remained in the resulting celloidin cast. By intrapelvic injections of dyes, air, and opaque media, in both post-mortem specimens and living animals, these authors came to the conclusion that the essential factor in this backflow is a minute trauma, from pressure, of the delicate pelvic mucosa at



FIG. 334.—The so called pyelovenous backflow, with lymphatic resorption in addition

the *fornix calycis*. Surrounding each fornix is a thin-walled venous plexus, into which the interlobar veins of the kidney drain, and the tissue separating the calycal and venous lumina is at this point very thin, delicate, and easily ruptured. In post-mortem injections the medium readily escapes into the interlobar, arcuate, and interlobular veins, but in the living subject the normal venous blood-stream prevents this retrograde flow, and the drug is seen moving towards the renal veins. According to *McAlpine*, some of the drug probably escapes also into the sinus renalis and lies in the fat of the hilum.

The practical applications of the above phenomena are two—strictly to avoid undue pressure during the pyelographic injections, and to use only non-toxic reagents. In this respect air must be regarded as in the dangerous class, since air embolus might result. This is possibly the explanation of sudden death following on air-distension of the bladder prior to cystostomy.

THE NEUROMUSCULAR APPARATUS OF THE KIDNEY AND URETER

The calyces and pelves of the kidney and the ureter together constitute a hollow muscular viscus exhibiting rhythmic systole and diastole, and subject to double autonomic innervation. The motor nerve supply comes from the vagi and reaches the kidney and ureter via the renal periarterial plexus. The inhibitory supply comes from the sympathetic, and is said to be derived from the eleventh and twelfth dorsal and first and second lumbar roots.

The muscular system of the kidney is complicated. The renal pelves and ureter have three muscular layers, according to *Jona*. At certain points the circular fibres are condensed into ring muscles, the ring-muscle system of the kidney and ureter described by *Kelly* and *Burnham*.

These ring muscles are situated as follows :

(1) **ROUND THE RENAL PAPILLÆ.**—This was described by *Muschat* as the *musculus spiralis papillæ*, and is said by him to exert a milking action of the papillæ, and so to fill the secondary calyces.

(2) **SPHINCTERIC RINGS** round the necks of the primary calyces, or calyco-pelvic junctions, and at the pelviureteric junction.

(3) **IN THE URETER** there are said to be two sphincters separating three ureteric spindles. The evidence for these is not so definite, but is supported by the varying calibre of the ureter, as follows : Pelviureteral junction 3 mm., lumbar segment 8 mm., upper sphincteric segment 4 mm., pelvic segment 6 mm., lower sphincteric segment 2 mm.

The Normal Peristaltic Cycle in the Kidney.—This has been studied by *J. L. Jona* by pyeloscopic and manometric methods. The pyeloscopic, which falls within the ambit of this book, merits description, since it is of in the study of dysfunction of the renal peristaltic activity. To study peristalsis pyeloscopically, it is necessary to fill the renal pelves from intravenous urography does not cast a sufficiently dense shadow. *Jo*

the intravenous preparation of Uroselectan B. or Per-Abrodil. This gives an exceptionally dense shadow which can be seen in the fluorescent screen. The renal pelvis is first drained as far as possible by aspiration, and then filled with the opaque injection. The subsequent emptying of the calycal system, according to the above worker, follows a definite orderly peristaltic sequence.

(1) The upper primary calyx (the term is meant to include the secondary calyces also) empties itself into the pelvis. Immediately this act has been completed, the sphincteric ring muscle at its neck contracts, and shuts off the calyx from the pelvis, thus preventing any reflux. This sequence of events occupies one to three seconds.

(2) After an interval of two to three seconds, the middle primary calyx undergoes the same sequence.

(3) Then, after the same interval, the lower primary calyx contracts. No sphincteric occlusion occurs at its neck, however, since almost immediately the renal pelvis contracts with a snap and ejects about $\frac{1}{2}$ c.c. of urino into the upper ureter, whence it is passed down from spindle to spindle into the bladder.

While these two lower calyces and the renal pelvis have been emptying, the upper has been refilling, ready to start the cycle again.

It should be noted that normally the emptying of the kidney lumen is very incomplete; a "maintenance filling" of about 8 c.c. is retained.

According to *Leguen*, the average rate of evacuation is rather slower than the above suggests, viz. 1 c.c. per minute. He states that a kidney which has been subjected to retrograde pyelography will usually clear itself of its opaque contents in about ten minutes.

Pregnancy normally causes some increase in the size and diminished tone of the kidney pelvis and abdominal ureter, from the fifth month onwards. It usually enlarges by about two-thirds of the normal but may go on to the stage of a mild hydronephrosis (Figs 335, 336). This is held by *Schumacher* to be due to pressure on the lower ureter, but *Jona* holds it to be due to the influence of an excess of œstrin. In support of this he cites the similar result of the injection of œstrin into nonparous mice. The ureter not only dilates, but also elongates, so that its course becomes slightly tortuous. This tortuosity normally disappears in time, but may persist in cases of subinvolution of the uterus.

Tambour tracings of the intrapelvic pressure in some cases give an interesting graphic record of intrarenal systole. In this the main rise in the curve is due to the pelvic systole, and the three ripples on the down curve result from the calycal systoles.

Neuromuscular Dysfunction.—According to the above authority, numerous forms of dysfunction may occur, the principal amongst which are (1) spasm of the whole pelvis, (2) atony of the whole pelvis, (3) spasm of the ureter at one point, with dilatation and antiperistalsis above, and (4) atony of the ureter. The cause of these dysfunctions is often quite obscure, but the spastic

phenomena are frequently reflex to a local pyelitis or ureteritis, or to an inflammatory lesion in the gall-bladder, appendix, or adnexa.

Renal Sympathicotonus.—Of the dysfunctions involving combined dilatation and spasm, the best defined is that described by *S. H. Harris*, under the name of *renal sympathicotonus*. He defines this condition as a dysfunction of the ring-muscle system, in which an achalasia of those sphincters results in renal stasis and eventual hydronephrosis.

The outstanding clinical features of this condition are that it is commonly unilateral, renal pain and costo-vertebral tenderness may be present, urinary stasis may occur, and there is occasionally an exaggerated knee jerk on the affected side.

Eserine gr. 1/60th hypodermically relieves the pain and by relaxing the spasm abolishes the urinary stasis. Both effects are temporary.

Radiographic Features.—Three stages of renal sympathicotonus may be shown by urography.

(1) **SYSTOLIC.**—This, the stage of irritability, shows a contracted pelvis and calyces, and delayed emptying when tested by the instrumental method. Eserine empties the pelvis and relieves the pain.

(2) **DIASTOLIC.**—The renal and calycal musculature seems to be exhausted, and to have given way before the pelviureteric spasm. The renal pelvis is therefore enlarged. Eserine again contracts it and relieves the pain.

(3) **DILATATION.**—The musculature is paralysed, and any degree of hydronephrosis may be present. Eserine relieves the pain, but does not contract the renal pelvis.

This condition is analogous to Hirschsprung's disease and cardiospasm, and is relieved by periarterial renal sympathectomy.

Action of Drugs on Dysfunction of the Ring-muscle System.—*Jona* has reported the following pharmacological reactions, as recorded by the kymographic tracings. In each case the drug was given hypodermically or intramuscularly.

PITUITRIN.—This causes an accentuation of the normal rhythm, or, in cases of atony and spasm, a return to the normal. It is a valuable agent in some cases, since, if it successfully breaks the abnormal cycle, the effect is lasting.

ATROPINE, HISTAMINE, AND ESERINE all cause a rise in the intrapelvic pressure. The similar effect of atropine and eserine is curious, in view of their opposite effects on the vagus.

QUININE causes a relaxation of the sphincter system, and is of value in spastic cases.

ASPIRIN by the mouth slows the rhythm.

Much of the above work on the neuromuscular apparatus is of recent date and further investigation may modify these views. In the meantime presents an interesting field for further research.

DEVELOPMENTAL ABNORMALITIES OF THE KIDNEY

Reference has already been made to the numerous developmental variations which lie within the limits of anatomical normality. Certain variations, however, may become pathological, or assume prime significance in the surgical treatment of pathological conditions.

Development of the Kidney and Ureter.—The rudiments of the kidneys appear about the beginning of the second month of intra-uterine life. Each arises as a diverticulum from the hind end of the Wolffian duct, close to where the latter opens into the cloaca. This diverticulum grows upwards into the intermediate cell mass. Its blind end dilates and subdivides to form the pelvis, calyces, and collecting tubules. The secretory tubules are formed from the primitive metanephros, and the connective tissue and blood-vessels from the mesodermic tissue. At first the kidney is in the sacral region and obtains its primitive blood-supply from the iliac artery or lower end of the abdominal aorta. It gradually ascends to its adult side in the loin, and there obtains a second and permanent blood-supply from the aorta higher up—the renal arteries. Normally, the primitive blood-supply disappears, but occasionally it persists as a supernumerary renal artery. This may arise from the common iliac artery; more commonly it comes from the aorta, and may be either superior or inferior polar. In the latter case it runs upwards and outwards to the lower pole of the kidney, in front of or behind the ureter. It is accounted by some as one cause of hydronephrosis, but opinion is still sharply divided on this point. It is certain that division of the vessel does not cure the hydronephrosis in many cases, and also that in many the dilatation does *not* begin immediately above the point at which the artery holds up the ureter. *Joseph* records a case of infantile hydronephrosis which he states was due to an aberrant artery. The truth would appear to be that obstructive inferior polar artery almost always primarily affects the lower part of the pelvis itself, and that the hooking up of the ureter is a secondary effect due to distension of the pelvis downwards past the obstruction.

FUSED KIDNEYS

There are several varieties of this anomaly. The commonest is the horseshoe deformity. Five different investigators, *Botez*, *Carlier*, *Thompson*, *Motzfeld*, and *Jack*, all find an incidence of 1 in 700 post-mortems. Males are more commonly affected than females, in a ratio of 8 : 3.

As the embryonic kidneys ascend and rotate into their adult position in the loin, they normally remain quite separate. Should they come into contact and adhere, a horseshoe kidney results, the two organs being joined by an isthmus, which lies in front of the spine, aorta, and inferior vena cava. In the majority of cases this bridge is formed of renal tissue, and joins the lower poles in front of the fourth and fifth lumbar vertebrae.

Fused kidneys are always low in position, and may be actually pre-sacral.

The upper poles are farther apart than the lower—in contradistinction to the normal—and as the normal developmental rotation of the kidneys cannot take place, the renal pelvis lie in front of the U-shaped renal mass. instead of medial to the two kidneys. The major calyces tend to look backwards, or backwards and inwards. The malrotation may affect one kidney only. the other assuming a fairly normal plane.

The ureters pass downwards and forwards in front of the isthmus, and tend to curve outwards in their upper portions, together forming the "flower-vase" contour.

The above is the common arrangement, but in 10 per cent. of cases the upper poles may be fused. In these cases the adrenals may be fused also. *Thompson* has recorded two cases of a solitary ureter draining two pelvises. *Ravasini* records one in which the ureter passed behind the isthmus. The ureters may be complete and bilateral, or incomplete, or absent on one side. The pelvises may show all variations between complete fusion and complete independence, and are commonly considerably deformed.

Radiographic Features of Horseshoe Kidney.—Plain radiograms may hint at the condition, from the absence of clearly defined renal outlines at the isthmus. *Köhler* has enumerated the following signs of horseshoe kidney, visible in a plain radiogram.

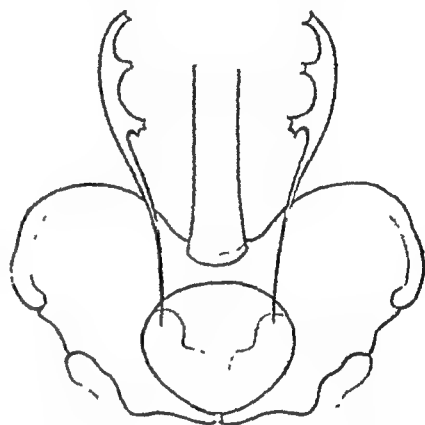


FIG. 337.—The "flower-vase" outline and the inturned calyces in horseshoe kidney (inferior polar fusion).

- (1) The long axes of the kidneys are parallel, and do not diverge below.
- (2) The kidneys are unduly close together.
- (3) Double and equal ptosis.
- (4) Immobility in the transverse direction.
- (5) Visible isthmus.

None of these is certain, however, and urography affords the only definite evidence.

The classical urographic signs are :

- (1) **FAILURE OF ROTATION OF KIDNEY.**—The calyces face inwards, or inwards and backwards.
- (2) **ECTOPIC POSITION OF THE KIDNEY.**—lumbar or sacral.
- (3) **POSITION OF THE URETER,** which enters the renal pelvis from anterior outer aspect. The two ureters curving outwards as they descend form the so-called "flower-vase" contour.
- (4) **THE LOWER PRIMARY CALYCES** may extend towards one another in inferior pole fusions.

(5) THE PELTS are unduly close together, and are larger than normal. Actual hydronephrosis is not uncommon.

These last two signs, calyceal and pelvic, have been systematised by



FIG 338 Hydronephrosis in the right half of a horseshoe kidney (proved at operation)



FIG 339 -Ascending pyelogram of the left half of a horseshoe kidney (same case as Fig 338) Note the intorted calyces and the line of the ureter, which with that of the other side, produces the "flower vase" contour.

Gutierrez into what he describes as the pyelographic triangle and its minimal basal angle.

This, an inverted isosceles triangle, is marked out as follows. The mid-point of the intercristal line forms the downward-pointing apex. The base is formed by a transverse line drawn at the level of the disc between the second

and third lumbar vertebræ. The points on this baseline from which the sides of the triangle are drawn (and the angle so mapped out) are arrived at by dropping perpendiculars from it to the innermost parts of the lower calyces. The inferior angle is the basal angle described by *Gutierrez*, and in the normal varies between 63 degrees and 100 degrees, with an average of 90 degrees.

In nineteen cases of horseshoe kidney recorded by him the average angle was 20 degrees. Other observers obtained a much higher figure than this, and the sign is not a very reliable one.

Rarer types of fused kidneys are the **discoid** and the **thyroid**, so named from their shape. The most important point in these cases is to determine that the

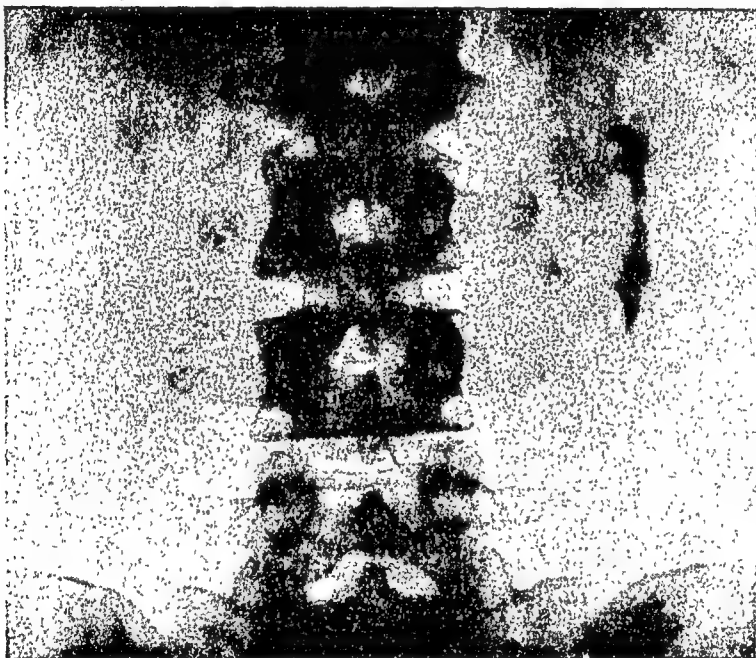


FIG. 340.—The same case as Figs. 338 and 339 after intravenous urography. The right hydronephrotic kidney was not visualised.

kidneys are fused, lest disaster occur by excision of the fused organ. It is also desirable to determine the independence or otherwise of the two halves, especially with reference to their vascular and ureteric arrangements. In view of the numerous possibilities, each case must be investigated on its merits.

Unilateral Fused Kidney.—In this type the fused kidney has passed to one side during the ascent. The homolateral kidney is above the other and is usually larger. This state of affairs will be at once apparent on instrumental pyelography, the ureter crossing over to the opposite side and there enlarging into a renal pelvis. The calyces tend to point to the midline in both kidney homo- and contra-lateral. If the two groups face in opposite directions, "S-shaped" or "sigmoid" kidney results.

OTHER CONGENITAL ABNORMALITIES OF THE KIDNEY

Congenital Absence of one Kidney.—It is similarly of vital importance to detect this abnormality. Cystoscopic appearances will raise its suspicion, and urography will confirm it.

Ectopic Kidney.—This condition results from failure, partial or complete



FIG 341. —A deformed ectopic hydronephrotic right kidney. The left renal pelvis and calyces are also deformed.

on the part of the kidney to ascend to its normal position in the loin. All stages between the adult lumbar and foetal sacral positions may occur. A few cases are recorded where it has ascended above the normal site. The left

kidney is more commonly affected than the right. Crossed ectopia is rare, other than in the type described under fused unilateral kidney.

The ectopic kidney is smaller than the normal, and is deformed, the degree of deformity running *pari passu* with the severity of the ectopia (Fig. 341). The pelves and calyces share in this deformity, and in severe cases the hilum faces forwards. The ureter is short and runs straight down to the bladder.

Instrumental pyelography is the method of choice, and demonstrates the following distinguishing features of the condition. (1) The ectopic position, (2)



FIG. 342.—Duplex right kidney, with double ureter in the upper third : intravenous urogram.

The fixation of the kidney in different postures, (3) The short ureter, and (4) The deformed pelvis. The last three distinguish ectopia from nephroptosis

Non-rotation of the Kidney.—In this the kidney is seen more or less edge on in an antero-posterior view, with a corresponding distortion and foreshortening of the pyelographic appearance.

Duplex Kidney.—In this common condition, when there is also a double ureter, only one half, the one into which the catheter has passed, may be demonstrated by an instrumental pyelogram. Usually the cephalic half is demonstrated. It presents a dumb-bell appearance and usually serves the upper third of the kidney. If the ureter be double for only part of its

the other half of the kidney will be filled if the injection is made through the partially withdrawn catheter. Intravenous pyelography does not suffer from this defect of the instrumental method, and should be used if there is reason to suspect a duplex kidney, and the other ureter cannot be located (Fig. 342). It is essential to determine whether the undemonstrated calyx has been obliterated by disease, or only "missed" by the examination.

Renal Hypoplasia.—In this condition the kidney is functionless, due to failure in development of the renal epithelium from the metanephros. The kidney is represented by a small mass of fatty and fibrous tissue, with perhaps some epithelial elements and small cysts. In some cases the ureter and pelvis develop, and in others these also are hypoplastic. In the former case instrumental pyelography reveals a rounded saccular pelvis, with no calyces. In the latter pyelography is impossible.

The type in which a pelvis and ureter are present may be distinguished from hydronephrosis by its small atypical shape and by the absence of calyces. It may, however, be confused with a dilated unicalycal pelvis in a case of duplex kidney and double ureter if the catheter has entered and filled that half only. Intravenous pyelography will differentiate between them, or injection through a partially withdrawn catheter.

Floating and Movable Kidney.—A kidney is said to be floating when it is provided with a peritoneal mesentery—a mesonephros. This is a rare congenital variation, and its very existence is denied by some authorities on the grounds that it is developmentally impossible. Movable kidney, a much commoner condition, moves up and down and laterally within the lax perirenal fascia. The renal vessels are elongated to allow of this movement, and the perirenal fat atrophied.

The condition can usually be detected clinically. Radiographic confirmation depends on the pyelogram. This shows the kidney to be variable in its position. If a radiogram be taken with the kidney displaced downwards, the upper part of the ureter is seen to be curved or looped on itself.

This looping of the upper ureter is the distinguishing point between a low mobile kidney and a low ectopic kidney. In the latter there is no "slack" in the ureter.

INFLAMMATION OF THE KIDNEY

Pyelitis.—The majority of cases of chronic pyelitis show no X-ray or pyelographic signs. Occasionally, if severe or long-standing, some clubbing of the calyces may be present, *the pelvis remaining of normal size*. At a late stage there may occur obstruction at the pelviureteric junction with secondary dilatation of the pelvis.¹ The ureter may be involved and become rigid and dilated, and subsequently stenosed.

Pyonephrosis. The later stage of the above merges into a pyonephrosis. In this the pyelogram shows calyceal and pelvic dilatation, with an irregular

¹ Clinically, however temporary blockage is frequent even in acute cases.

woolly outline to the calyces. The woolliness is due to ulceration and granulation issue. The contrast medium may extend into the renal parenchyma, indicative of a medullary or cortical abscess communicating with the calyx. Such a track must be distinguished from the normal filling of the collecting tubules after instrumental pyelography with Uroselectan B. A pyonephrosis may also be secondary to a hydronephrosis, from super added infection, either ascending or hæmatogenous. It is frequently associated with renal calculus. The radiographic appearances are either those of hydronephrosis, or if much ulceration be present, of the pyelonephritic hydronephrosis described above. In the acutely painful periods there is complete blockage of the ureter, and no pyuria: in this phase the intravenous method commonly fails to demonstrate the condition.

Perirenal Abscess.—This condition may give several radiological signs of its presence. The *diaphragm* commonly shows reflex fixation on the same side, and, if the abscess is large, some elevation. When the stage of elevation is reached, the abscess has probably spread to a subphrenic type. The *renal outline* may be obliterated, enlarged, or displaced. The shadow of the *psoas muscle* is not infrequently obliterated by a perinephritic abscess, and the *lumbar spine* may show a mild scoliosis, the concavity towards the abscess. The *colon*, if sufficiently outlined with gas, may show some displacement.

In acute cases instrumental and intravenous urography is inadvisable, but in the more chronic cases, amenable to such investigation, associated pathological conditions may be demonstrable, such as pyonephrosis, defective renal function, pressure deformity of the pelvis or calyces (Fig. 343), or a fistula between kidney and abscess.



FIG. 343.—Large retrorenal abscess displacing the kidney and ureter forwards and inwards and causing a pressure defect in the lower calyx. Complete recovery followed drainage of the abscess

RENAL AND PERIRENAL FISTULÆ

Radiography usually gives accurate information as to the connections of these fistulæ.

(1) **Fistulæ unconnected with the Urinary Apparatus.**—Such fistulæ may arise from an empyema, appendicitis, or other purulent collection. The proof of its relationships may be made by combining pyelography with injection of the fistulous track with lipiodol. The former will show the urinary apparatus to be intact, and the latter the course of the track. It is important to employ stereoscopy in this. Only by stereograms can the spatial relationships be clearly made out. In addition, the mouth of the fistula should be marked with a wire ring fixed on the skin by adhesive strapping. This strapping also serves to seal the sinus, and prevent leakage of the contrast medium during the exposures.

(2) **Fistulæ connected with the Kidney.**—These are rarely spontaneous, from the rupture of a pyonephrosis into the perinephric tissue and thence on to the surface of the body. In such cases it may be sufficient to employ trans-vesical pyelography alone, and as soon as the medium commences to leak at the mouth of the fistula, to seal the latter with strapping, inject a little more of the medium, and take stereoscopic radiograms. If this procedure is ineffective, an attempt may be made to fill the fistula and renal pelvis from the loin or to use both methods.

PYELO-URETERITIS CYSTICA

Bieberbach, Cook, and Goodale have reported the X-ray appearances in a case. The left kidney and ureter were involved.

Pathologically the mucosa of the renal pelvis and ureter was studded with translucent cysts, 2-3 mm. in diameter. These cysts are supposed to be post-inflammatory, and to result from central degeneration in hyperplastic mucosa. (*Brunn.*)

Radiographically slight hydronephrosis and hydroureter were present, and characteristic tiny round filling-defects in the pyelographic shadow. These were best seen in the ureter.

As this appearance can be simulated by bubbles of gas introduced during an instrumental pyelogram, it is advisable to reinject and take a further radiogram, should the first show such an appearance.

TUBERCULOSIS OF THE KIDNEY AND URETER

Pathology.—This infection may attack the kidney in the following forms :

(1) **MILIARY TUBERCULOSIS**—This cannot be demonstrated radiographically.

(2) **ULCERO-CAVERNOUS TYPE.**—This is the common form and commences with ulceration of the apex of a pyramid. The ulceration gradually erodes and hollows out the pyramid. A similar process occurs in other pyramids and by coalescing forms considerable cavities.

(3) **TUBERCULOUS PYO- OR HYDRONEPHROSIS.**—Contracture of the necks of the ulcerated calyces may shut off the cavity and produce a partial hydro-

nephrosis, or the ureter may become similarly contracted at some part of its course (usually the lower end) and cause a total hydronephrosis with a hydroureter.

(4) CASEOUS TUBERCULOSIS.—When the infection is chronic, massive caseation may take place, and the whole kidney may eventually be transformed into a compartmented sac filled with caseous masses. Finally calcification takes place.

Radiographic Features.

ULCERO-CAVERNOUS TYPE.—This may accurately be demonstrated by pyelography. Without question the instrumental method is much to be preferred, as it is essential to obtain the finest details of the calyceal outline. The cystoscopy necessary for this also allows the outflow from the other kidney to be investigated, a matter which is of the greatest importance in determining whether nephrectomy is advisable.

The earliest lesion is a "moth-eaten" enlargement of one of the calyces. Later a number of calyces show the same change. By coalescence of these, larger irregular cavities are formed, until the disease develops into the stage of tuberculous hydronephrosis. In the early stages the ureter loses its "spindles," and becomes dilated and atonic (Fig. 344).

TUBERCULOUS PYO- OR HYDRONEPHROSIS.—The distinguishing feature of this is the irregular "moth-eaten" outline, in contradistinction to the clear-cut contour of simple hydronephrosis. The ureter, if affected, may show in a series of radiograms to be irregularly dilated and rigid, with intervening strictures (Fig. 345). A feature is the constancy of the ureteral irregularity in this stage. If it also becomes shortened, as it frequently does, the ureter loses its normal curves and runs straight from the renal hilum to the bladder, which also shows distortion.

CASEOUS TUBERCULOSIS.—In neither of the above types does plain radio



FIG. 344.—Ultero-cavernous tuberculosis of the left kidney and ureter with almost complete destruction of the parenchyma. The sole symptom was pyuria, discovered at life-insurance examination. On cystoscopy a grossly distorted bladder was seen, and a tuberculous ulcer at a "golf-hole" left ureteric orifice.

graphy afford much help, but in the caseous type quite characteristic signs are given in a plain radiogram. The renal outline may be somewhat enlarged, and scattered throughout the renal areas are faint, hazy, woolly opacities, irregularly arranged in clumps, and representing the caseous masses (Fig. 346). A pyelogram of this type shows an irregular "spider" type of pelvis, with little in the



FIG. 345 —Tuberculous hydronephrosis and hydroureter with structure of the latter at its lower end (Ascending pyelogram)

way of calycal dilatation. In the stage of calcification the woolly shadows become denser and more clear-cut, until finally the kidney may shrink and become an irregularly calcified mass, almost resembling a huge calcified lymphatic gland.

While the instrumental method of pyelography is greatly to be preferred in the investigation of renal tuberculosis, because of the clarity of the outline so obtained, it is frequently impracticable. It is precluded in cases in which the

bladder is severely involved, or where the degree of involvement of the ureter makes ureteric catheterisation impossible. If there be grave mixed tuberculous and septic infection present, the method is likewise contraindicated. In such cases it is wiser to be content with the less certain information afforded by the intravenous method. The latter has the advantage that it gives some informa-

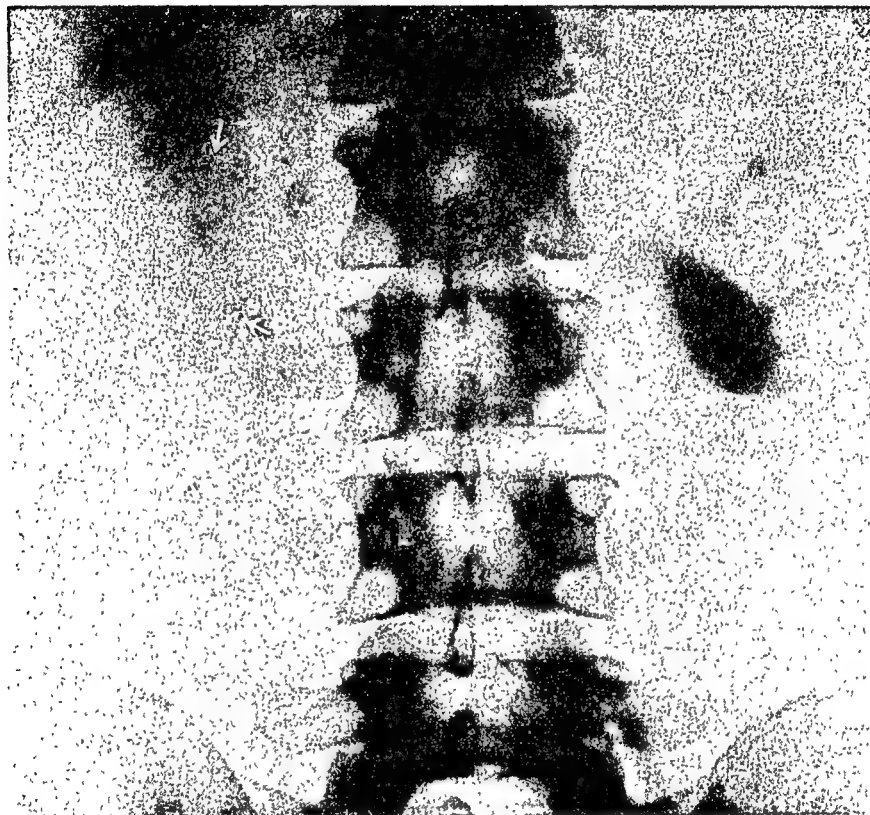


FIG. 346.—Caseous tuberculosis of the right kidney, showing caseous and calcareous debris : intravenous urogram.

tion as to the function of the other kidney in unilateral lesions, or may show an unsuspected lesion in it : of importance where surgery is contemplated.

CYSTS OF THE KIDNEY

Polycystic Kidney (Congenital Cystic Kidney)

CLINICAL FEATURES.—In this condition the kidney contains a large number of cysts ; the enlargement, often very great, produces a uniform lobulated tumour ; and occasionally the kidney has the appearance almost of a bunch of grapes. It is supposed by some to result from a failure to unite on the part of the collecting tubules, developed from the primitive ureteral bud, and



FIG 347.—Polycystic disease of the kidneys

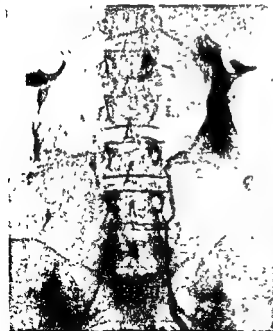


FIG 348.—Polycystic disease of the kidneys, showing the enlargement of the pelvis, the spider calyces, and the crescentic impressions produced by the cysts

secreting tubules, developed from the metanephros. The latter, ending blindly, dilate into cysts. There are, however, objections to this view, and there are numerous other theories of its causation, including proliferating cyst-adenoma, lymphangioma, endothelioma, Wolffian inclusions, and foetal nephritis. The condition is frequently familial. It may be present at birth, or may not be fully developed until adult life. It is always bilateral, and causes an enlargement of the kidneys, that can usually be detected clinically. There

being few other bilateral enlargements of the kidney, the disease is usually strongly suspected on clinical grounds.

RADIOGRAPHIC FEATURES.—Lobulation of the renal outlines may be visible in a plain radiogram, but this is an inconstant sign. Pyelography usually gives a very characteristic appearance. Owing to the defective urinary excretion frequently present in these cases, the intravenous method is likely to be unsuccessful in outlining the renal pelvis satisfactorily, and the instrumental method should be used if possible.

As the cysts, varying in size up to that of a cherry, are scattered irregularly throughout the renal substance, the distortion which results in the pelvis and calyces may be very varied. Three

features in the pyelogram can generally be made out (Figs. 347-50).

(1) General enlargement, lengthening and compression deformity of the pelvis and calyces, of the "spider" or "dragon" type. The primary calyces are more involved in this change than the secondary.

(2) Absence of any localised hydronephrotic dilatations.

(3) The calyces and pelvis may here and there show clear-cut crescentic indentations, sometimes with a "Half-shadow," due to the projection of the cyst into the calycal or pelvic lumen. This is the most characteristic sign, and, if well developed, is pathognomonic of polycystic disease.

These changes are bilateral, a point which differentiates the condition from neoplasm.

Solitary Cyst.—Solitary serous cysts are very rare. They may become very large. In a pyelogram they may cause pressure distortion and elongation of one part of the pelvis and associated calyces (Fig. 351). The wall of the cyst may undergo calcification.

Hydatid Cysts.—These are also very rare in Great Britain, only four cases having been reported. They are less uncommon in Russia, Greece, Spain, and Australia.



FIG. 349.—Congenital cystic kidney. This pyelogram shows the characteristic features: marked enlargement of the cavity of the kidney, lengthening of the calyces and crescentic indentations produced by the pressure of the cysts.

R. C. Begg points out that there are three classes of hydatid cyst of the kidney—open, closed, and pseudo-closed, according to the relation of the cyst to the renal pelvis. Each presents different radiological characteristics. In the *open type* the pericystic and endocystic cavities both communicate with the renal cavity, and the cyst therefore becomes filled with the opaque medium



FIG. 330.—Polycystic disease of both kidneys, and a large hypernephroma of the lower pole of the left. Many small metastases were present in the liver

in pyelography. The filling of the cyst may be irregular, owing to the presence of endocystic daughter cysts. Renal colic and the passage of hydatid elements in the urine are typical features in this type.

In the *closed type* both cyst and pericyst are completely shut off, and cause

a pressure filling-defect in a pyelogram. The symptoms in these cases are those of pressure of the cyst, if the latter is large.

In the *pseudo-closed type* the renal pelvis communicates with the pericystic space. The pyelographic medium may escape into this space, and cause an irregular shadow round the cyst.

Calcification of the wall of these cysts is not uncommon, and appears in a radiogram as a more or less complete shadow.

In contradistinction to hydatid disease of the liver, primary hydatid in the kidney (i.e. derived from a hexacanth embryo) is almost invariably single, and from this secondary cysts may develop.

Secondary embolic cysts are rare in the kidney, 25 in 474 cases (*Nicaise*). Their behaviour is similar to the primary type.

Aneurysm of the Renal Parenchyma.—This is a rare condition, and becomes visible in a plain radiogram if its wall is calcified. It then shows as an irregular circular shadow, with a coarse network or series of irregular shadows from calcification in the anterior and posterior walls.

Some indentation deformity of calyces and pelvis may result and be visible in a pyelogram.



FIG. 351.—Filling defect in a pyelogram due to a simple cyst of the kidney.

RENAL NEOPLASMS

The classification and macroscopic pathology of renal neoplasms is of importance, inasmuch as their site of incidence and local spread permit

cases of individual radiological identification. The following classification includes most of the neoplasms met with in the kidney.

There have to be distinguished tumours of the

- (1) Renal parenchyma.
- (2) Renal pelvis.
- (3) Renal capsule.
- (4) Adrenals.
- (5) Perirenal tissue.
- (6) Prerenal tissues.

SIMPLE NEOPLASMS OF THE RENAL PARENCHYMA

Simple tumours such as lipoma in combination with other tissues (e.g. lipomyoma, angiolipoma), fibroma, angioma, and fibromyoma are common, but are usually of no clinical significance. An exception to this is when hæmaturia originates from an angioma of the renal pyramid or pelvis. These tumours are usually quite small.

Adenoma is a rare tumour and seldom reaches a larger size than a walnut. In a case observed by one of the writers (*C. J. M.*) the tumour attained a diameter of 3 inches, and caused a semicircular defect of the lower half of the pelvis and an obstruction of the upper calyces. The clinical features were globular enlargement of the kidney and pain from degeneration of the tumour. There was no hæmaturia. A point of note is that even if these simple tumours do grow to a size sufficient to deform the renal pelvis, they do not produce the spider deformity so characteristic of hypernephroma (Fig. 352).

MALIGNANT NEOPLASMS OF THE RENAL PARENCHYMA

These form the majority of renal neoplasms and include hypernephroma, teratoid malignant tumours, carcinoma, and sarcoma.

Hypernephroma is much the commonest malignant tumour of the



FIG. 352.—Simple polycystic tumour of lower pole of kidney, causing pain in loin. Removed by wedge resection of lower pole. Kidney elsewhere healthy.

renal parenchyma. This striking tumour was described by *Grawitz* in 1883, and concerning its pathogenesis—its origin as an adrenal inclusion, or from the Wolffian body—controversy still continues. The clinical and pathological features of hypernephromata are of great radiological importance. They most

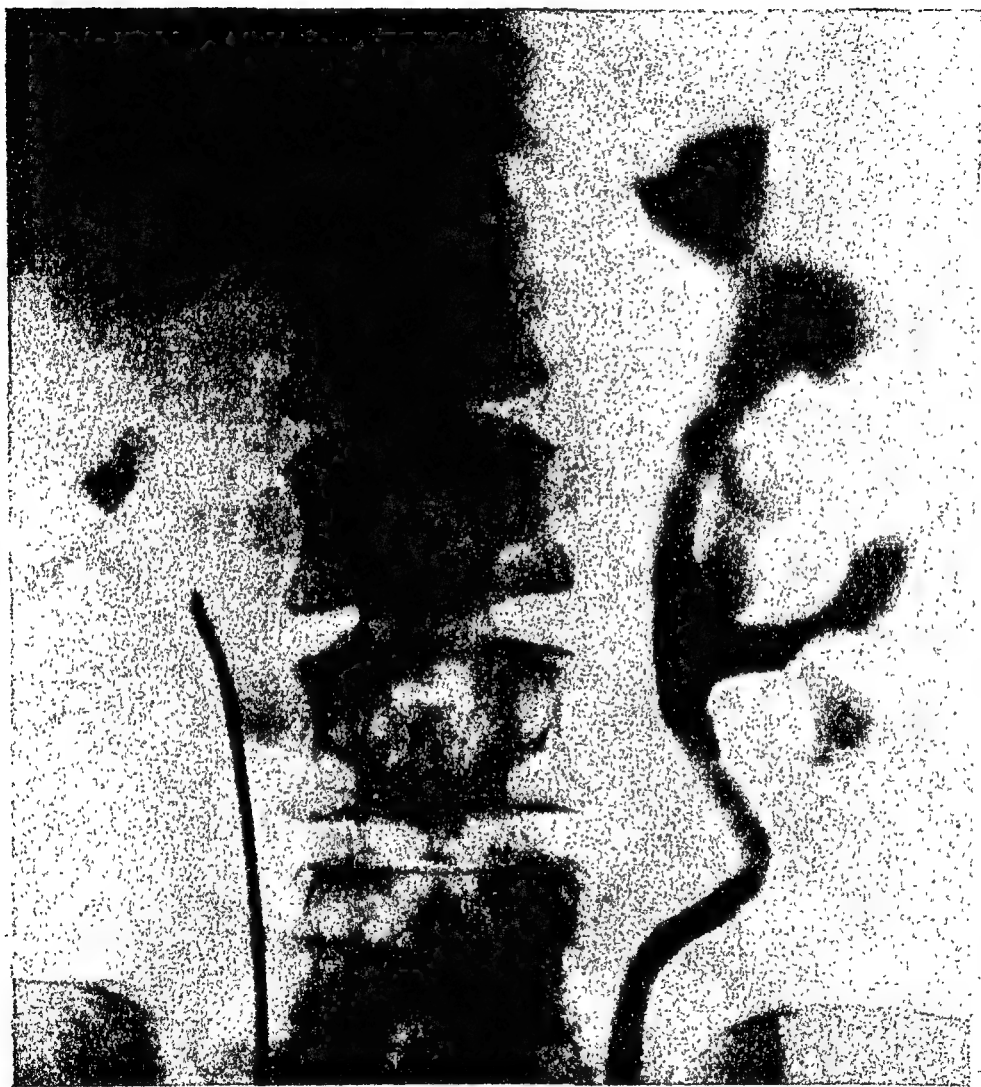


FIG. 353.—Hypernephroma of the left kidney.

frequently arise in the cortex of the upper pole, near the capsule, cause an irregular enlargement of the renal outline, and also encroach on the lumen of the kidney. Hæmaturia is the common symptom, as a result of extension of a calyx. Early metastasis is the rule, both by lymphatics and blood-stream. Often there occurs a very early and extensive hæmatogenous spread to

lungs and bones—cranial vault, sternum, and spine—so frequently that examination of these structures is imperative in cases of suspected or proved hypernephroma, and particularly before nephrectomy is decided upon. An obvious



FIG. 354.—Filling defect in the left kidney, caused by a hypernephroma of the upper pole.

corollary is that a careful search should be made for hypernephroma in case of unexplained pulmonary or bone metastases.

Radiographic Features of Hypernephroma.—It may be possible to demonstrate in a plain radiogram the irregular enlargement of the renal contour, but since the enlargement is usually in the upper pole it may not be clearly defined. Perirenal emphysema after the method of Carelli shows the upper pole clearly, and is an available measure in exceptional cases in which pyelography fails.

Calcification is a rare occurrence in a hypernephroma, and is, of course, visible in a plain radiogram.

Apart from the above, the radiological signs of hypernephroma are all pyelographic (Figs. 353-355). *Brausch* enumerates these as follows:

- (1) Elongation of calyces.
- (2) Encroachment of the pelvis.
- (3) Secondary pyelectasis.
- (4) Displacement of the kidney and its pelvis.
- (5) Deformity of the pelviureteric junction and upper ureter.

(1) *Elongation of Calyces*.—Elongation of a calyx is the earliest sign of the majority of malignant parenchymal tumours. It is especially well marked in the hypernephromas. As the tumour expands the kidney substance outwards, the calyx is stretched out with it. At the same time it is narrowed by compression, deformed, perhaps displaced, and the terminal papillary expansion deformed or obliterated. As the tumour grows, the adjacent calyces become involved. To this deformity the terms "spider" pelvis, "dragon" pelvis, and "spider-leg" deformity have been applied. The appearance must be distinguished from anatomical abnormalities. Sometimes a healthy pelvis may show a long spidery calyx. The secondary calyces are, however, normal in such cases, and the appearance may be the same in the other kidney.

(2) *ENCROACHMENT ON RENAL PELVIS*.—As the tumour extends towards the kidney pelvis, it encroaches progressively on its lumen. In the early stages a small filling-defect may result; in more advanced cases only irregular narrow clefts may remain or the lumen may be completely obliterated. In this last case, the injected medium is arrested at the pelviureteral junction. Carcinoma shows this feature more than the other renal neoplasms, but it also occurs in hypernephroma.

(3) *SECONDARY PYELECTASIS*.—Rarely the tumour may encroach on and obstruct the pelviureteral region, and produce a secondary pyelectasis. Or it may occlude the isthmus of a primary calyx and cause a localised dilatation of that calyx. It may not be possible to demonstrate this with instrumental pyelography, and in such a case the intravenous method may be of value.



FIG. 355.—Hypernephroma of the upper pole of the right kidney, giving an appearance of amputation of the upper calyx. The growth extended into the renal vein. The only symptom was hæmaturia one month before.



FIG 356—Barium enema pressure deformity of the sigmoid from a large left renal tumour

(4) **DISPLACEMENT OF THE RENAL PELVIS.**—This occurs when the tumour is large. The direction of displacement of the renal pelvis depends on its spatial relationship to the growing tumour mass.

(5) **DEFORMITY AND DISPLACEMENT OF THE UPPER URETER.**—This also occurs when the growth is a large one.

If the renal tumour is very large, it may cause pressure deformity of other viscera, such as stomach or colon (Figs. 356–357).

Teratoid Tumours are rare, they occur in infants; and may attain a great size, with little tendency to metastasise locally or systemically. Hæmaturia is not a feature of the disease, as a rule. Carcinoma or sarcoma may predominate in the histological picture, and a variety of other tissues may be included, such

as plain or striped muscle, cartilage, fatty and elastic tissue, and epithelium.

The radiographic signs are similar to those of hypernephroma; enlargement of the renal outline, calyceal deformity and obstruction, and compression of the renal pelvis

Carcinoma of the Kidney (pace that section of pathological opinion that calls all hypernephromata carcinoma) differs from the latter in that the kidney is invaded slowly, and not distended or displaced, as in the case of hypernephroma. The tumour arises from the renal tubules and does not attain a great size. In the early stages invasion of the renal pelvis occurs, with an



FIG 357—Displacement of stomach by a large left renal tumour.

irregular filling-defect. Later, increasing infiltration may cause calycal deformity and local or general calycal hydronephrosis. The typical appearance in carcinoma of the renal parenchyma is thus different from that of hypernephroma, but the appearances in both may be so varied that the X-ray evidence will not be adequate in most cases to differentiate between them.

Sarcoma of the Kidney (if a true sarcoma—most prove on close examination to be teratomata) is commonly subcapsular or perirenal in origin, and tends to spread outwards. This enlarges and obscures the renal contour, but pyelographic changes are either absent or slight and late in appearance.

NEOPLASMS OF THE RENAL PELVIS

Papilloma.—This is a comparatively rare condition. It causes a filling-defect in the pelvis in a pyelogram, and, depending on the amount of obstruction at the pelviureteral junction, may induce a hydronephrosis. To demonstrate the filling-defect, radiograms should be made with different degrees of distension of the pelvis by the opaque injection.

The type of filling-defect depends on the shape and size of the tumour mass. The smaller ones may produce a discrete rounded or nodular filling-defect. A large villous growth produces an irregular spotty or lacework defect, the result of the contrast fluid occupying the interstices of the growth. In the true renal pelvic tumour the calyces are intact, and fill to normal contours with transvesical pyelography, but portions of a villous growth may block a calycal neck, and prevent it filling with the medium.

A bubble of gas, accidentally injected along with the contrast medium into



FIG. 358.—Hæmorrhage into the pelvis and ureter, causing filling in the pyelogram, from blood-clots.

the renal pelvis, can cause a filling-defect closely similar to that of a small pedunculated tumour, and must be excluded in the differential diagnosis. Similarly, a blood-clot in the renal pelvis may cause a suspicious filling-defect, which can be differentiated only by subsequent re-examination (Fig. 358).

Epithelioma.—In its papillary form this presents similar radiographic features to the above. When an appearance of this nature is obtained, therefore, suspicion of the graver condition should always be entertained. Rarely epithelioma may occur in a squamous form.

When, in carcinoma of the renal pelvis, the obstructive hydronephrosis becomes very marked, it may be impossible to show it either by the instrumental or intravenous method, the medium in the former case being unable to enter the kidney, and in the latter not being concentrated by it adequately for demonstration.

Mesoblastic Tumours of the renal pelvis (*fibroma, sarcoma*) are very rare, and their pyelographic appearances have not been recorded sufficiently often to form a collected picture

NEOPLASMS OF THE RENAL CAPSULE

Fibroma and *sarcoma* may arise from this site and cause an irregular enlargement of the renal outline. The renal pelvis is not as a rule deformed.

NEOPLASMS OF THE PERIRENAL TISSUES

Among such are *fibro-myxo*, or *angioliopoma*, and *liposarcoma*. The kidney may be displaced or rotated, without change in the pyelogram

ADRENAL NEOPLASMS

These come but little into urological or urographic diagnosis. In the cortical variety the well-known virilism syndrome calls attention to the condition. There are two medullary varieties the *Hutchinson* type with orbital metastasis and resulting exophthalmos, and the *Pepper* type, with large hepatic secondaries. In the latter the tumour is commonly palpable, and a pyelogram may show compression of the upper calyces and depression of the kidney. *Hæmaturia* is not a feature.

PRERENAL TUMOURS

These may be hydatid cysts, lymphangiomata, or developmental cysts of Wolffian or Mullerian origin. Sometimes the tumour may be of the Grawitz type, completely distinct from the kidney. Indeed, similar tumours may be found in the inguinal canal, testis, and broad ligament, all situations in which rests may occur.

All the above prerenal tumours may cause displacement of the kidney without pyelographic changes.

HYDRONEPHROSIS

For a proper appreciation of the radiographic features of hydronephrosis some understanding of the ætiology, pathology, and morbid anatomy is necessary. It may be defined as a chronic aseptic dilatation of the renal basin and calyces. In the majority of cases it is to be regarded as symptomatic of a preceding pathological condition; in certain cases, however, it is the hydronephrosis itself which constitutes the main picture.

ÆTIOLOGY

The causes of hydronephrosis are many, and may be grouped according to whether it is bilateral or unilateral.

Bilateral Hydronephrosis arises from chronic vesical obstructions, namely:

(1) From urethral stricture—traumatic, inflammatory, developmental, or neoplastic.

(2) From bladderneck obstructions—prostatic enlargement and fibrosis (simple or malignant), median bar, middle lobe, and contracted bladderneck.

(3) In the "spinal bladder," from spinal cord lesions—traumatic, vascular, neoplastic, inflammatory, tabetic, and idiopathic (in 20 per cent. of cases no nerve lesions can be demonstrated).

(4) In congenital achalasia of the bladderneck, associated with Hirschsprung's disease. In this connection it is interesting that in some cases of variously disturbed vesical innervation in children, radiography may reveal a spina bifida occulta, suggesting cord pressure.

(6) In bilateral ureteral obstruction, from carcinoma cervicis and some conditions of vesical fibrosis (e.g. bilharziasis) and in the latter stages of pregnancy.

In all the above types the hydronephrosis is accompanied by bilateral hydroureter.

Unilateral Hydronephrosis is due in general to intermittent or constant partial obstruction in the ureter or at the pelviureteric junction.

URETERAL OBSTRUCTION may occur anywhere along its course from obstruction of the lumen by calculus or tumour, or changes in the ureteral wall from œdema, ulceration, or scarring, following trauma or impaction of a calculus (Fig. 359).

A not uncommon type of this is fibrosis at the lower end from tuberculosis, producing a tuberculous hydronephrosis (*vide* Fig. 345).

Obstruction may be caused by pressure from without from tumour or fibrosis (Fig. 360). The problematic pressure from aberrant artery has been discussed under developmental abnormalities of the kidney (Fig. 361). It is a dubious cause in many cases, as is kinking of the ureter from nephroptosis.

All the above unilateral types of hydronephrosis are accompanied by hydroureter above the point of obstruction.

OBSTRUCTION AT THE PELVIURETERAL JUNCTION may occur from various causes. It may be due to stone, or more commonly the associated fibrosis; to

tumours of the renal pelvis, such as papilloma or carcinoma; to fibrosis in chronic mild pyelitis. Most commonly, however, no pathological change is present at the pelviureteral junction. Such cases in all probability belong to



FIG. 359.—Double calculous hydronephrosis, shown by instrumental pyelography. A small calculus in the left kidney has been blotted out by the opaque medium. One large coraliform calculus and two small ones are visible on the right side.

hydroureter, a bladder hypertrophied to a thickness of 1 cm., the ureter was not obstructed.

the great group of achalasias which include pharyngeal diverticulum, cardio-spasm, and Hirschsprung's disease. High origin of the ureter is a frequently quoted cause, but it is debatable whether the high origin is not really secondary to an achalasia hydronephrosis.

Congenital Hydro-nephrosis, occurring at or soon after birth, may be due to a variety of causes. If *unilateral*, it may be due to stenosis of the ureter, or folds or kinks in it. The commoner type is *bilateral*. In one variety it is due to urethral obstruction, usually at the meatus. The one most likely to be met with is the achalasia type. In one such case (C. J. M.), there was, in an infant dying at nine days after bilateral hydronephrosis and

PATHOLOGICAL ANATOMY

In the early stages of hydronephrosis the pelvis increases in capacity, the calyceal necks widen, the calyces enlarge, and the pyramids become first flattened and then hollowed out. The kidney is not appreciably enlarged at first.

In the developed stages the hydronephrosis takes one of two forms, the renal (or calycal) and the pelvic.

In the renal type the organ becomes grossly hollowed out, its size increased,



FIG. 360.—Mild hydronephrosis from adhesions of the perirenal fascia.



FIG. 361.—Developed hydronephrosis associated with an inferior polar artery.

and the pelvis small and opened out. The end stage is a sacculated bag. In this variety the external renal contour may show lobulations.

In the pelvic type the dilatation is at the expense of the renal pelvis. The calyces are but slightly dilated, and the kidney sits like a cap on the globular pelvis. In this type the angulation of the pelviureteral junction is most

It is said to develop a valve action, and so promote a vicious cycle. The ureter is seldom dilated in this type.

Where for any reason a calycal neck or one segment of a double pelvis becomes obstructed, a *partial* hydronephrosis, confined to the calyces affected, results. This is a very important variety from the point of view of differential diagnosis.

RADIOGRAPHIC FEATURES

Little or nothing regarding a hydronephrosis can be learnt from a plain radiogram. Renal or pelvic enlargement may be made out, but not the cause



FIG 362 —Double hydronephrosis: the left shown by ascending pyelography. Note the catheter coiled in the right renal pelvis, indicating that it too is dilated.

thereof. The clearest demonstration of the degree and type of hydronephrosis is given by the instrumental method of contrast urography.

With Instrumental Pyelography the earliest indication of a hydronephrosis is a slight clubbing of the secondary calyces. The calyces dilate somewhat, their cupping tends to be lost, and the necks become thicker. The renal pelvis at the same time begins to enlarge (Fig. 360). The late X-ray appearances depend on which type hydronephrosis develops.

IN THE CALYCAL TYPE the calyces undergo progressive enlargement, until the intervening renal parenchyma is reduced to thin septa separating large regularly arranged loculi (Figs. 359, 361, and 362). The pelvis also dilates, but not as a sac external to the kidney. Its concave infero-medial border becomes convex, and an angulation appears between it and the ureter.

IN THE PELVIC TYPE the predominant feature is the large bulbous pelvis, contrasted with the bunch of slightly enlarged calyces arranged along its outer margin. As the pelvis enlarges it tends to rotate the kidney behind it, so that the renal hilum points forward. The kidney is then seen in a pyelogram more or less edge on, and the pelvic shadow is superimposed on the calycal (Fig. 364). In the renal or calycal type this rotation does not take place.

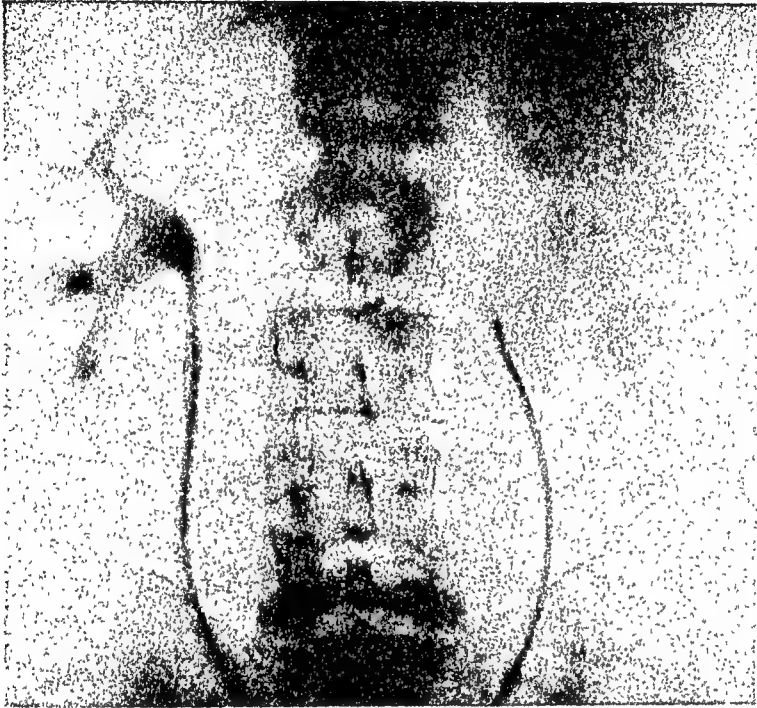


FIG. 363.—Huge left hydronephrosis, shown by ascending pyelography.

Indeed, a reverse rotation tends to occur, and the kidney assumes a coronal plane. The pyelogram is thus seen undistorted by any obliquity.

In a case of PARTIAL HYDRONEPHROSIS of one or more calyces, these will be seen to be dilated in an instrumental pyelogram, provided that the causal obstruction is not too tight to allow the contrast medium into the dilated portion. Some clue as to the cause of the obstruction, e.g. tumour, may also be evident.

It may, of course, be added that catheterisation of the obstructed kidney may in itself immediately give clear proof of abnormal capacity.

Intravenous Urography finds one of its most useful spheres in the demonstration of hydronephrosis. The back pressure always present in these cases promotes complete filling, and in unilateral cases the other kidney affords a normal standard against which to judge the diseased organ. The venous method also gives a fairly accurate indication of the function of

affected kidney. The X-ray appearances are usually quite definite, especially in a unilateral case (Fig. 365).

If early, the affected kidney secretes the drug-laden urine only slightly more slowly than the normal one, but if a suitable series of radiograms be taken in the first ten minutes after the injection a slight lag can be made out. The drug is slower in appearing, and the contrast poorer. At the same time the more



FIG 364 —Marked pelvic hydronephrosis, associated with an inferior polar artery

faintly contrasted calyces and pelvis are seen to be rather larger. This particularly applies to the calycal necks, which are so commonly contracted down to a narrow channel in the normal, but remain wide in the hydronephrotic kidney. In other words, their normal peristaltic contractility is in abeyance. These two features, then—slight dilatation and lag in secretion—present in the early hydronephrosis, become more marked in the advanced case, while the third change, lack of contrast, becomes prominent.

In the advanced hydronephrosis of the pelvic type the lag in secretion may be quite slight, and the calyces early be seen in good contrast. This is because the kidney substance is not grossly damaged. The dilated pelvis, however, already contains a considerable quantity of urine, and so it may be some time—up to twenty to thirty minutes—before the concentration of the drug is high enough in the saccular pelvis to throw a dense shadow. Frequently all that is seen is a large, rather faint shadow, through which may be seen the densely filled calyces, the drug-containing urine in them being undiluted. In gross

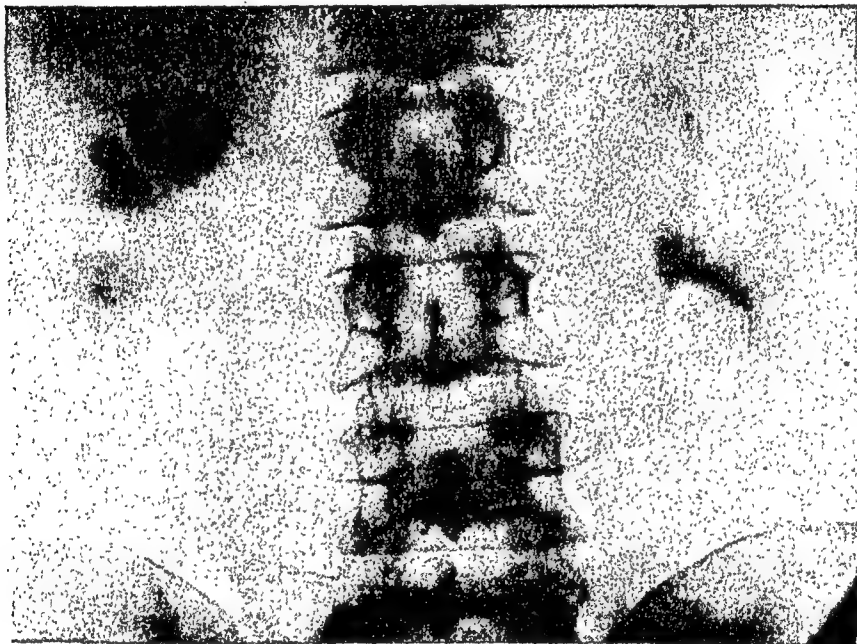


FIG. 365.—Calycal hydronephrosis, showing the characteristic “tulip” appearance (intravenous urogram).

cases the intravenous method fails entirely and the instrumental procedure is indicated.

In the renal type of advanced hydronephrosis rather a different appearance is seen with intravenous urography. As the renal tissue is partially destroyed, secretion of the drug is poor and consequently the density of the shadow also. The most marked feature is, however, the lag in the secretion. In a developed case of double hydronephrosis and hydroureter the best shadow may not be seen for as long as three to four hours (Fig. 366). In cases, therefore, where hydronephrosis of this type is suspected on clinical grounds and no shadow appears in the first hour, it is as well to re-examine the patient at subsequent hours, in the hope of finding a shadow later. As a rule, however, the lag is of such a degree as to cause the optimum concentration to appear a half to one hour after the injection.

While the intravenous method therefore does not give a pyelographic shadow equal in density to the transvesical, it very frequently shows enough accurately



FIG. 368 —Uroselectan B urogram, 60 minutes after injection, showing bilateral hydronephrosis and hydroureter in a case of fibrous enlargement of the prostate

to indicate the morbid anatomy, and in addition affords such valuable evidence regarding function as to make it the method of first choice *in this condition*, the instrumental method being reserved for those in which the intravenous fails.

It must be added that there are some cases in which both methods of pyelography fail to demonstrate the renal distension, e.g. the first or "systolic" stage of renal sympathicotonus (*q.v.*).

URINARY CALCULUS

Calculus is the commonest condition for which X-ray examination of the urinary tract is requested. The radiogram forms much the most important piece of evidence in the diagnosis of that abnormality.

Ætiology.—A calculus is a progressive conglomeration of crystalloid bodies bound together by a cement substance. The former are precipitated out of the urine; the latter, in which they reach saturation by adsorption, is provided by the plasma in the form of an irreversible colloid (such as fibrin). Bacterial infection commonly produces a *nucleus* of colloid, which is infiltrated by crystals; where infection has resulted in *urinary alkalisation*, simple chemical precipitation of triple phosphates occurs without the intervention of a colloid framework, and the calculi are of a much looser amorphous texture.

Chemical Composition.—The radiographic demonstration of a calculus depends on its opacity to X-rays. This in turn depends, apart from the size and porosity of the stone, primarily on its chemical constitution; more precisely, on the atomic number of the elements composing it. The form of the chemical combination of these elements is of no importance in this respect. The higher the atomic number, the greater the opacity of the element to X-rays.

Below is a list of some of the relevant atomic numbers, together with those used in contrast media, etc.:

H. 1	Mg. 12	Br. 35
C. 6	P. 15	Ag. 47
N. 7	S. 16	I. 53
O. 8	K. 19	Ba. 56
Na. 9	Ca. 20	Pb. 83

The main bulk of the tissues of the body, excluding bone, is composed chiefly of hydrogen, oxygen, nitrogen, and carbon, with an opacity to X-rays equal approximately to that of water. A body in the tissues, such as bone or calculus, containing calcium, phosphorus, magnesium, or sodium in any quantity, will cast a shadow in a radiogram.

Urinary calculi are rarely formed of a single salt. As a rule a nucleus is present, surrounded by laminae of varying composition.

The nucleus usually consists of ammonium urates in infants, uric acid in young adults, and calcium oxalate in calculi commencing after the age of 40.

The superadded laminae may be composed of the following: uric acid ammonium and sodium urate, calcium oxalate, calcium phosphate, calcium

carbonate, ammonium and magnesium phosphate, cystin, xanthin, indigo blood. Calcium oxalate is the most frequent component, accounting for near half; uric acid is the next.

VARIETIES OF CALCULI

Calcium Oxalate Calculi.—These are usually single, and rarely attain size larger than a filbert. They have a mulberry or sometimes finely spiculate outline. They may be laminated. They cast the densest shadow of all the calculi, because of the large amount of calcium they contain.

Phosphatic Calculi, either calcium phosphate, or more commonly triphosphate of calcium, ammonium, and magnesium, are usually secondary, i.e. are superimposed on a nuclear stone in infected alkaline or ammoniacal urine. This applies particularly to the triple phosphate type. They tend to grow quickly, and often attain a great size. They typically assume a dendritic or coralliform shape, forming a cast of the renal cavity. This cast may be one or several faceted pieces. Lamellation is common, and the radiographic shadow is usually very dense.

Urate Calculi.—These are usually a mixture of one or more of the following: ammonium, sodium, calcium, and magnesium urate. The stones are usually small, rounded, and smooth. They occur particularly in children. The shadow cast by them is of a moderate or feeble density. Lamellation is common.

Calcium Carbonate Calculi.—These seldom occur in the pure state. Most commonly calcium carbonate is present in the layers of a triple phosphate stone. When pure, they cast a very dense shadow (cf. calcareous glands).

Uric Acid Calculus is a rare type, but an important one as, if it is pure, it casts no shadow. As a rule, however, it is mixed with urates, and then shows some opacity in a radiogram. These calculi are often laminated. They do not attain a large size.

Cystin, Xanthin, and Indigo are extremely uncommon types of recurrent calculi. If pure, they cast no shadow. If mixed, the opacity they cause is due to the calcium they contain (and sulphur in the case of cystin), and they usually contain so little that only a very faint opacity is visible in a radiogram.

The percentage of radiographically invisible calculi has been variously estimated at from 10 to 20 per cent. This figure varies somewhat, according to the degree of perfection of technique.

SITUATION AND NUMBER OF CALCULI

Situation.—A single renal calculus may occupy a position in the pelvis, calyces, or parenchyma. The pelvis is much the commonest site. The parenchymatous situation is relatively rare, and a calculus there is generally symptomless and only accidentally discovered.

When multiple phosphatic stones are present, some frequently occupy the calyces.

Number.—Calculi, especially the phosphatic, are frequently multiple, and in 20 per cent. of cases bilateral.

RADIOGRAPHIC DIAGNOSIS OF RENAL CALCULUS

This is concerned with these questions :

- (a) If a shadow is present, is it intra- or extrarenal ?
- (b) If it is intrarenal, is it due to a calculus ?
- (c) If no shadow is evident, is an invisible calculus present ?

The evidence is—

A. Intrinsic Characters

There are certain characteristics which if present indicate a shadow to be cast by a renal calculus. Some of these, such as coralliform shape, are



FIG. 367.—Small stone in the lower calyx of the left kidney.

pathognomonic; others are less definite. The characteristics may be classed as follows (Figs. 367–369) :

(1) **SITE OF THE OPACITY.**—It must be within the renal-pelvic outline in each plane. The renal outline is usually clearly visible; not so the pelvic, and its position must be estimated by that of the kidney hilum.

In the lateral view the renal outline cannot be made out, but the shadow of a renal calculus shows through that of the lumbar spine.

(2) **THE SHAPE OF THE OPACITY.**—This varies according to the type of stone. The oxalate calculi are mulberry or rounded, as are the uratic stones. The phosphatic are angular or coralliform.

(3) **THE TEXTURE OF THE OPACITY.**—The texture is either homogeneous or



FIG 358—Coralliform calculi in both kidneys



FIG 359—Huge right renal calculus, and many small calyceal ones

laminated. Stippling of the shadow, so typical of a calcified gland, is rarely seen in a calculus.

(4) **THE MOVEMENTS OF THE SHADOW**—The shadow should show the same respiratory shift as the kidney. This is an uncertain sign, and, so much more

certain are other available methods of diagnosis is rarely if ever worth the trouble of estimating it.

B. Urographic Characters

If the plain radiogram fails to give unequivocal evidence, either intravenous or instrumental urography will settle the problem (Figs. 370-373). When calyces or renal pelvis are outlined, their relationship to the doubtful shadow is clear. The contrast medium, if of feeble density, envelops the shadow, which can still be seen. More commonly the shadow is simply blotted out by the medium. If doubt still exists after an antero-posterior pyelogram, a lateral view will finally settle the site of the shadow. This nearly always also settles the *cause* of the shadow, as the causes, other than calculus, of a dense intrarenal opacity are very few—calcified tubercle, calcified neoplasm, and possibly teratoma.¹

The third diagnostic question—is there an invisible calculus present?—can rarely be settled by radiology except in the unusual case where a transparent stone causes a “negative” shadow. Even then it may be impossible to differentiate it from a neoplastic filling-defect. This uncertainty does not, however, occur in cases of invisible obstructive calculus in the ureter, in which the hold-up or hold-down of the contrast medium constitutes a reliable sign.

DIFFERENTIAL DIAGNOSIS OF RENAL CALCULUS

A large number of structures other than calculi can cast a shadow in or near the renal area. Most of these have characteristics which enable them to be distinguished.

Calcified Lymphatic Glands.—These form the commonest source of possible error in diagnosis. Such a mistake should seldom be made. The shadow of a calcified gland is typically mottled and granular; its texture is not uniform, and its outline is woolly and irregular. The collection of granules forming the shadow are usually rounded. If the calcified glands are in the mesentery, they usually show mobility, and do not remain constant in site in a series of radiograms. In all doubtful cases, pyelography settles the issue.

Cholelithiasis.—Calcified gall-stones cast a shadow in the right renal region in an antero-posterior radiogram. The ringed shadows of multiple stones cause no difficulty. The calcified “solitaire” may. The lateral view, pyelography, and cholecystography are the methods by which to differentiate the two, in that order of adoption (Fig. 373).

Pancreatic Calculi.—These are very rare. A single one may closely simulate a renal calculus, but several are usually present, scattered throughout the gland,

¹ Urography affords striking evidence at times that not only is calculus present, but also that calculus is not the primary disease, and that stone is secondary to obstruction. The clearest indication is given, therefore, that removal of the stone or stones would be merely symptomatic treatment, and that relief of obstruction is an essential in therapy.

when their distribution differentiates them from renal calculi. If suspected, their relationship to the duodenum may be shown by a barium meal. Pyelography serves to differentiate them from renal calculi, unless their shadow coincides with that of the renal pelvis. In that case oblique or parallax views are necessary.

Calcified Costal Cartilages.—These commonly cast a shadow in the renal areas in a radiogram of the urinary tract. The shadows are dense and granular, and frequently outline completely or incompletely the lower costal cartilages anteriorly. Widespread calcifications leave no room for doubt as to their



FIG. 370.—Round stone in the lower calyx of the left kidney, proved by ascending pyelography, which blots out the stone, and shows a mild degree of hydronephrosis

nature. Even in the cases where there are only one or two localised areas involved, the long axis of the shadow, corresponding to that of the cartilage and the shift in position in angled views differentiate them from urinary calculi.

The Tips of the Transverse Processes of the upper lumbar vertebra are normally much denser in a radiogram than the remainder. This disparity is frequently very marked, and in radiograms which fall short of the highest standard only the tip may be visible. In such case it might be mistaken for a calculus in the renal pelvis or, more probably, in the ureter.

Intestinal Contents.—Enteroliths are always quoted in this respect, but they enter into the question practically. Much commoner and more impor-



FIG. 371.—Large stone in the right renal pelvis, and a calcified left abdominal gland.



FIG. 372.—Uroselectan B. urogram of the same case as Fig. 371.

tant is the bismuth- or barium-impregnated scybalon. Such are found multiple as a rule, and their texture and distribution along the colon

nature. Occasionally gas in the colon is so disposed as to cast a negative ring shadow round a centre of normal translucency. The latter may then, by contrast, be mistaken for a calculus. In both the above re-examination after further preparation settles the issue.

Tuberculous Calcification in the Kidney, Atheromatous Vessels, and Warty Nævi on the Back are rare causes of confusion, but are usually easily



FIG 373 —Gall stones (→) and stones in the right kidney, differentiated by intravenous urography

differentiated, the first two by their respective mottled and striated shadows, and the last by inspection of the skin.

Calcification of the Adrenal Gland may result from tuberculosis. It causes an irregular striated shadow, which may outline the adrenal body more or less completely, lying close to the body of the twelfth dorsal or first lumbar vertebra. Its relation to the upper pole of the kidney is a further distinguishing feature.

CHAPTER XL

THE URETERS

The Ureters, like the kidneys, present many variations in their anatomical relationships, and in their radiographic appearances.

The ureteral line is described as follows : It commences at the pelvi-ureteral junction near the tip of the transverse process of the second lumbar vertebra, and proceeds vertically downwards, taking the tips of the succeeding lumbar transverse processes as its course. Reaching the basin of the pelvis, it inclines inwards somewhat, and passes down just medial to the sacro-iliac joint. At the lower margin of that joint, it sweeps outwards and follows a curve about an inch inside the true brim of the pelvis, to end in the trigone of the bladder. The above description is the classical one, based on the radiographic appearance after opaque catheterisation. Intravenous urography has shown what a marked rectification of the ureteral line is produced by the ureteral catheter, how much the ureter normally bends and wanders in its course from kidney to bladder, and what little significance is to be attached to such bends, windings, and kinks *in the absence* of the signs of ureteral obstruction.

The Ureteral Calibre.—In order to assess the importance of any narrowing or dilatation in the ureter, due consideration must be given to the normal ureteral calibre, and to anatomical and physiological variations.

There are described in the ureter four points of anatomical and physiological constriction, with three intermediate enlargements or "spindles." The narrowings are at the uretero-pelvic junction, at the level of the pelvic brim, an inch or so short of the bladder, and finally in the intramural portion (Fig. 374). Of the spindles, the lumbar is the longest and most pronounced and the final one often absent. The average normal measurements of the ureteral calibre are : uretero-pelvic junction 3 mm., lumbar spindle 8 mm., lumbo-sacral sphincter 4 mm., pelvic spindle 6 mm., and pelvic sphincter 2 mm.

There is also considerable variation in the response of the ureter to instrumentation ; in some cases the ureter may be spastic on catheterisation. and in others it may remain lax and of wide patency.

The Ureteral Course is also subject to much variation. Tortuosity is often quite a feature. It is of no practical importance, as the transference of urine from kidney to bladder is a peristaltic act. If the tortuosity be marked, it may indicate descent of the kidney. A mild degree of tortuosity is inevitable on forced inspiration.

Kinking is also often seen. This may be apparent or real. It is more



FIG 374—Normal instrumental pyelogram of both kidneys, showing the ureteric spindles on the right side

usually visible at some stage. The upper spindle tends to act as a temporary reservoir for urine until enough has collected in it to stimulate a peristaltic wave. The constriction at the pelvic brim commonly results in a section of the ureter in this zone not being visualised, whilst above and below it the upper and middle spindles are clearly outlined

often only apparent, the result of a tortuous bend in the ureter being seen end on in the radiogram.

The above variations occur chiefly in the upper spindle, and depend on the position of the kidney, the amount of retroperitoneal fat, the formation of the perirenal fascia and of the ureteral fascial sheath.

The anatomy of the ureter is best demonstrated radiographically by instrumental pyelography. Rectification of the ureteral line by the catheter can be avoided by gradual withdrawal of the catheter during the injection, till only an inch or so remains in the ureter when the exposure is made

The functional activity of the ureter can be studied in serial intravenous urography. By this means the upper and middle spindles are

DEVELOPMENTAL ABNORMALITIES

Double Ureter, Partial or Complete, is much the commonest developmental error found in the ureter. It is of great importance that this condition should not remain undetected during instrumental pyelography, by catheterisation of only one half of the double system. All stages of bifid ureter, from a bifid renal

divis down to two complete ureters, each with a separate vesical ostium, may be found. The division is usually from above downwards and is more commonly partial. The ureters may, however, be double below and single, or even blind, above. To fill them both during instrumental pyelography, the catheter should be withdrawn as far as possible, a procedure that is effective in all save those in which the doubling is complete, when catheterisation of both ostia is necessary.

COMPLETE DOUBLE URETER ASSOCIATED WITH DUPLEX KIDNEY, usually is a very constant anatomical arrangement. The upper pelvis represents the upper major calyx, with one or two calyces. If only one is present, the "dumb-bell" appearance results. The lower pelvis represents the remaining two-thirds of the normal renal pelvis. The upper ureter in the majority of cases begins mesial to the lower, passes behind it to the outer side, and near the bladder crosses again behind the lower to end in a vesical ostium mesial and caudal to the ostium of the lower. This arrangement of the vesical ostia is so constant as to have earned the title on the Continent of the *Weigert-Mayer* law. It is of importance to the cystoscopist, and the arrangement is visible pyelographically. The importance of intravenous urography in the demonstration of cases of bifid and double ureter is obvious, owing to the possible failure to demonstrate the whole system by the instrumental method.

Unilateral double ureter is said to occur in 4 per cent. of individuals (*Thomson-Walker*). Bilateral double ureter is much rarer. As many as six ureters have been found in one individual, according to this writer.

Several very rare abnormalities remain to be noted, most of which are susceptible to either instrumental or intravenous demonstration.

Ectopia of the ureter may occur, the duct ending below in the urethra, the seminal vesicle, the uterus, the vagina, the vestibule, or the duct of Gaertner. Such ureters are often supernumerary.

The ureter may end blindly above or below, but only the former of these could be radiographically shown.

Varied ureteral abnormalities may be found in cases of renal fusion. The ureters may be entirely or partially fused, or quite separate.

OBSTRUCTION OF THE URETER

Obstruction of the ureter is usually easily demonstrable radiographically. When well developed it gives characteristic evidence of its existence in the form of hydrourter and hydronephrosis. While these are the only unequivocal radiographic signs, to demand this "trade-mark" before diagnosing ureteral obstruction clinically would be to exclude from that category a considerable number of cases of renal and ureteral colic in which no gross obstruction nor any calculus can be detected. Radiography can give positive evidence in some of these cases, in which such clinical diagnoses as "ureteritis," "ureterospasm,"

"nephritis dolorosa hæmorrhagica," or "nephralgia" are commonly made (*vide* neuromuscular mechanism of the kidney).

If the obstruction becomes complete, the kidney loses its function and it is impossible to demonstrate the condition by intravenous pyelography. The instrumental method will show the lower limit of the atresia.

The causes of ureteral obstruction are many. Some of these are demonstrable radiographically.

(a) **Impacted Stone.**

(b) **Stricture of the Ureter.**—This usually results from previous gross damage, such as impacted stone, pyelo-ureteritis, or tuberculous ulceration. It is said to occur in rare cases without previous trauma. Radiographically the dilated ureter is seen to narrow to a point at the site of the stricture.

It is of importance to differentiate between organic stricture and uretero-spasm. The latter condition is described in the section on the neuromuscular mechanism: also the use of pituitrin and quinine in differentiating the two.

(c) **Obstruction from an Aberrant Artery.**—In this the obstruction is high up, close to the pelvi-ureteric junction, and the ureteral angulation is abrupt. The artery may actually cross the lower part of the renal pelvis.

(d) **Kinking of the Ureter, from Mobile Kidney.**—This is the classic explanation of Dietl's crises, but probably occurs much less commonly than was previously supposed. Most of these cases show no pelvic obstruction and are due to torsion of the neurovascular renal pedicle. They should be studied by the intravenous method. No significance should be attached to the appearance of a ureteral kink, if there is no sign of dilatation above. In such a case the supposed kink is probably an anatomical bend in the ureter seen end on. In the true kink which is causing intermittent obstruction there should be evident a hydroureter above, an acute angulation of the ureter, and a filling-defect at the point of angulation.

(e) **Adherent Calcified Gland.**—This is an occasional cause of ureteral obstruction. The retroperitoneal lymphatic glands may lie in close relationship to the ureter, separated only by the fascia transversalis, which forms a sheath for the kidney and ureter. Before a gland can implicate the ureter, however, the sheath must be implicated, and this makes ureteral obstruction from this cause a rarity.

URETERAL CALCULUS

In the main, ureteral calculi are formed in the kidney. An exception is the alleged type which develops *de novo* in a dilated ureter immediately above a stricture. Therefore the varieties found are similar to those found in the kidney, with the limitation in size imposed by the ureteral lumen.

As in the case of renal calculus, two questions may present themselves:

(a) If a shadow is visible, is it due to a calculus?

(b) If no shadow is visible, is an "invisible calculus" present?

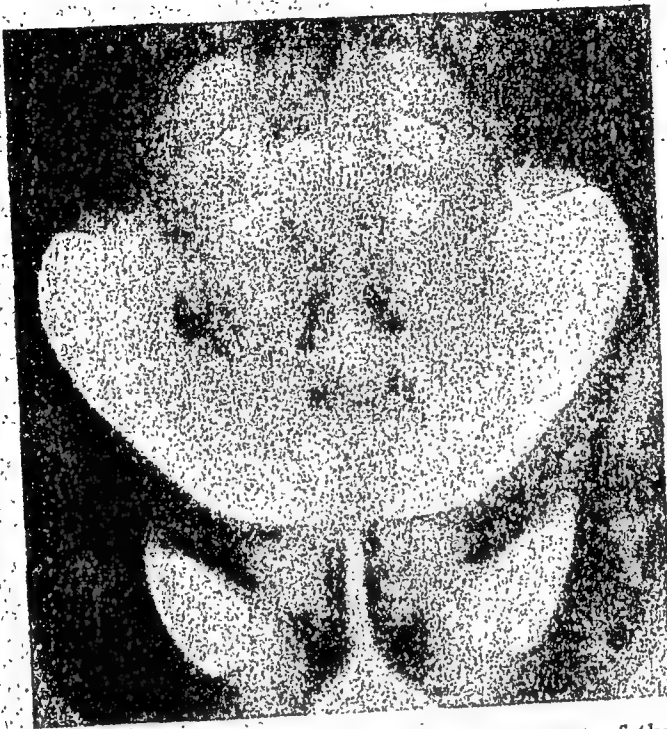


FIG. 375.—Calculus impacted in the lower part of the right ureter.

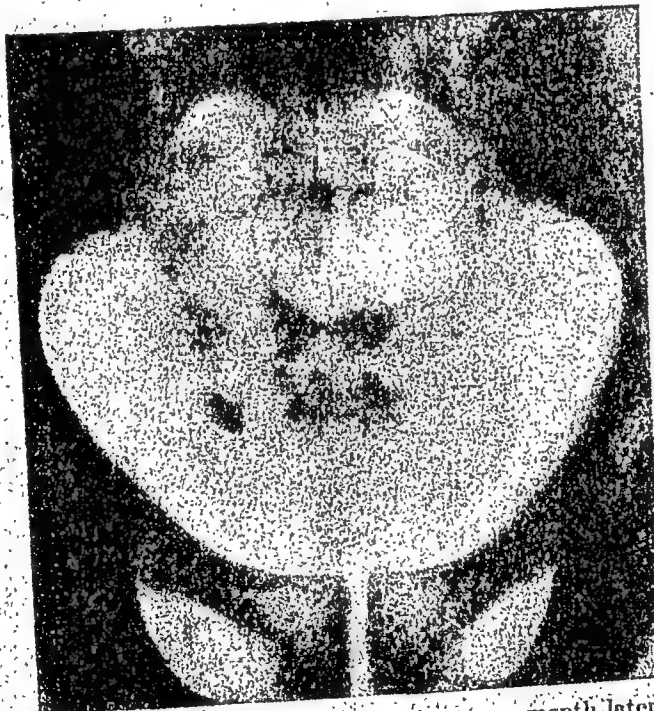


FIG. 376.—The same case as Fig. 375, one month later. The stone is near the lower end of the ureter.

In the case where a shadow is present, a positive diagnosis may often be made with fair confidence on the intrinsic appearance of the opacity, but certainty can often be attained only by demonstrating its spatial coincidence with the ureteral lumen.

A. Intrinsic Characters of Visible Ureteral Calculi

Ureteral calculi, if recently descended from the kidney, may be round, oval, or angular, and the surface may be smooth or crenated. Lamination is occasionally visible.

If the calculus has been impacted for some time in the ureter, it tends to become more oval, and if its sojourn there has been protracted, it may become considerably elongated in shape. The long axis of the stone is always in that of the ureter (Figs. 375-376). If a number of calculi are present, they may show a rosary effect in the line of the duct, a rare but characteristic appearance.

B. Differential Diagnosis of Visible Ureteral Calculus

Numerous radio-opacities may confuse the issue, as in the case of renal calculus.

CALCAREOUS LYMPHATIC GLANDS are the commonest, so far as the upper ureter is concerned. The mesenteric glands, the ileocolic, the lumbar, and the presacral may all cast a confusing shadow. Their characteristics have already been described in the section on renal calculus.

PHLEBOLITHS in the pelvic veins are often a cause of difficulty. They are present in a large percentage of individuals—33 per cent. according to Köhler. They are rounded, clear-cut in outline, homogeneous in texture, and frequently multiple. If multiple, they are, as a rule, bilateral and grouped in the region of the lower part of the ureter, on either side of the vesical neck. *Multiple phleboliths* are usually recognisable as such from their distribution, which clearly shows they cannot all be in the ureter; the chief danger in the interpretation of these shadows is to fail to recognise a small ureteric calculus in a group of phleboliths. *The solitary phlebolith* in the line of the ureter provides a problem which is frequently insoluble without urography, so closely may it simulate a calculus.

The method of Ziegler may help to differentiate between a calculus in the lowest part of the ureter and a phlebolith. This consists of a double exposure on one film, the bladder being filled between them. A calculus is displaced upwards by this procedure; a phlebolith is not. Contrast urography, however, affords much more accurate information, and Ziegler's method is rarely warranted.

OTHER CONDITIONS.—There are numerous other shadows which may rarely simulate a ureteral calculus. The tips of the lumbar transverse processes have already been mentioned. Others are calcareous arteries, calcified ascro-sciatic ligament, calcified fibroids, concretions in the vermiform appendix

r in colonic diverticula, and teeth in teratomata. Usually these opacities present characteristics which reveal their true nature, but in the doubtful case contrast urography must be used.

C. Urographic Diagnosis of Visible Ureteral Calculi

The demonstration that a given shadow is cast by a body in the ureter is effected by filling the lumen thereof with a contrast medium (either opaque

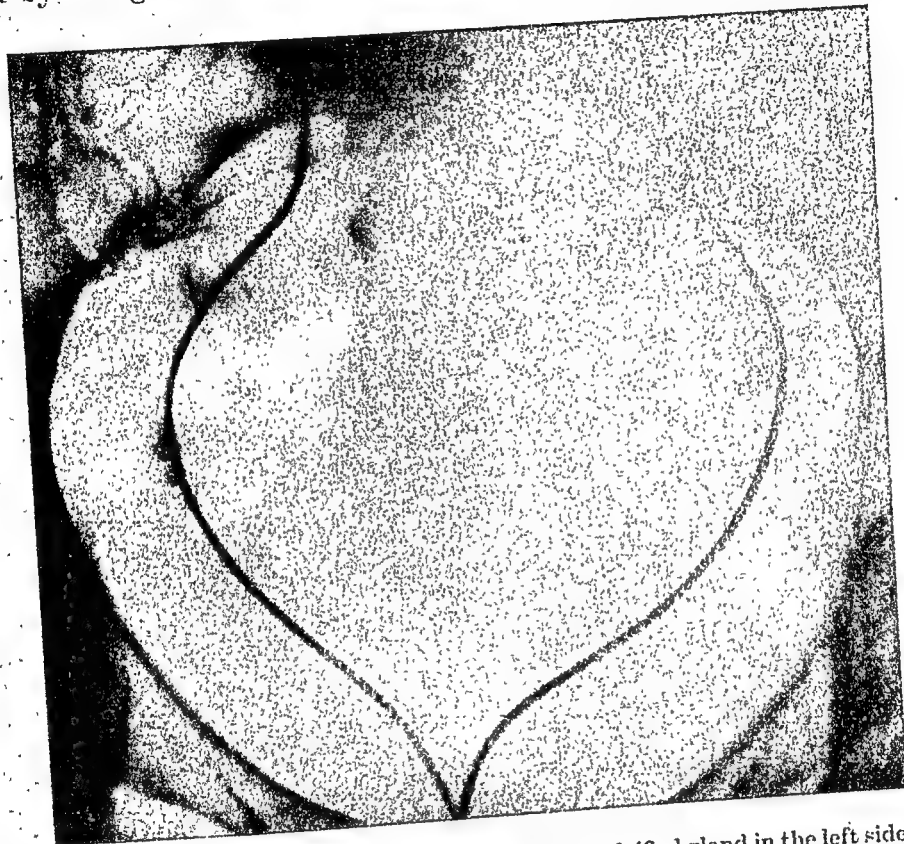


FIG. 377.—Four calculi in the right ureter and a calcified gland in the left side of the pelvis, both orientated by opaque catheters.

catheter, opaque injection, or intravenous urography) and proving the coincidence of the shadow and the now-evident lumen in *two planes* (Fig. 377). If only one plane be used, an opaque body might superimpose its shadow on that of the ureter and simulate a calculus. However, if a contrast medium be injected after passage of the catheter, ureteral dilatation above the shadow is likely to be seen if a stone is present. Ureteral dilatation and obstructive hydro-nephrosis above a suspected stone are usually conclusive evidence and are well demonstrated by the intravenous method (Figs. 378-379).

The parallactic principle is utilised in the method of *Kretschmer*, in



FIG 378 - Obstructive hydronephrosis of the right kidney from ureteral calculus, shown by excretion urography (30 minutes after)

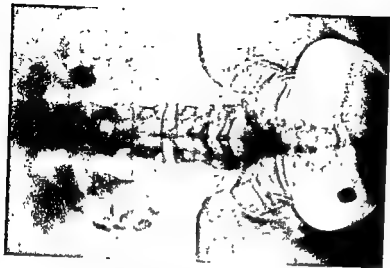


FIG 379 - Obstructive hydronephrosis of the right kidney following on impaction of a stone in the lower part of the right ureter. Intravenous urography (30 minutes after)

after the passage of an opaque ureteral catheter, two exposures are made on one film, the tube being shifted transversely 6-10 cm. between the exposures. The shift of catheter and calculus are the same. A gall-stone, on the other hand,



FIG. 380.—Stone impacted in the lower part of the right ureter, producing reflex anuria in the right kidney. Intravenous urography five minutes after injection.

would show a greater shift, being nearer to the source of irradiation than the ureteral catheter.

The method of *Pozzi* and *Proust* consists in displacing the stone upwards by the opaque catheter, between two successive radiographic exposures. It is not applicable to impacted stones.

While the above measures are available in difficult cases, straightforward urography in one plane is conclusive in the vast majority of cases.

The Invisible Ureteral Calculus.—This forms a problem only less difficult than its equivalent in the kidney. It is estimated that in about 20 per cent. of cases a ureteral calculus is transparent. The exact proportion depends on the quality of the radiograms and on the thoroughness of the clinical diagnosis: antagonistic factors, be it noted, which account for considerable variation in the published figures. The radiographic demonstration depends on whether there is any obstruction to the medium—intravenous or retrograde—caused by the stone (Fig. 380), and on the detection of a filling-defect in the instrumental ureterogram. The intravenous method shows only the stasis above in these



FIG 381 — Invisible " calculus in the lower part of the right ureter, causing obstruction to the catheter and injected opaque medium

cases; the filling-defect is not usually evident. According to *McAlpine*, in rare cases the stone may become impregnated by the injection, and so cause a half-shadow when the injection is in situ, and a faint opacity after evacuation.

Acute Impaction of a Ureteral Stone.—When a stone becomes suddenly impacted in a ureter—i.e. during the stage of acute renal colic—the ureter is spastic, and acute urinary stasis occurs in the kidney. *Helmer*, quoted by *Graham Hodgson*, has pointed out that intravenous injection of Uroselectan B. may produce a dense homogeneous opacity of the whole kidney in about five minutes. The opaque drug is dammed back in the renal cortex, and the calyces and renal pelvis are outlined in negative relief. This does not always occur, as in Fig. 380, where anuria resulted without opacity of the kidney.

URETEROCŒLE

(*Syn.* cystic dilatation of the lower end of the ureter. intravesical ureteral cyst.)

This consists of an intravesical cystic dilatation of the terminal portion of the ureteral mucosa, forming an inner layer, an outer (vesical) layer of vesical mucosa, and a varying amount of muscular and alveolar tissue between. The ureteral ostium is narrowed in some cases, and in others it is normal.

The ætiology is unknown. It is held by some to be the result of congenital malformation of the orifice. Acquired obstruction, ureteral atony, and congenital weakness of Waldeyer's sheath have all been mentioned as pathogenetic factors. It occurs equally in both sexes and at all ages. Calculus formation commonly occurs in the ureterocœle, but this is held to be a result and not a cause of the condition. Usually the diagnosis is clear on cystoscopy, but the condition may also be demonstrated by intravenous or retrograde urography.

Radiographic Features.—These vary, according to whether there is a calculus in the sac or not.

A. URETEROCŒLE CONTAINING A CALCULUS.—*In a plain radiogram* the calculus is visible at the region of the ureteric orifice and is fixed in that position. *On filling the bladder per urethram* with an opaque solution, the calculus is seen to be intravesical in all views: i.e. it is not in a diverticulum. Separating the calculus from the opaque fluid is a translucent halo cast by the wall of the ureterocœle. *With intravenous urography* the lower ureter becomes visible. It is usually dilated, and combined with the bulbous intravesical termination forms the "snake" ureteric shadow; so-called from its resemblance to a cobra.

B. URETEROCŒLE NOT CONTAINING A CALCULUS.—*The instrumental cystogram* reveals a rounded filling-defect corresponding in size, shape, and position to the cystic swelling. The appearance with *intravenous urography* depends on the stage of the examination. At first, before the bladder has filled, the cobra ureter will be in evidence. Later, when the bladder is sufficiently distended with Uroselectan-laden urine, the opaque cyst is demarcated from the filled bladder by a halo, as described above, but without the added density of the calculus (Fig. 382). Finally, after evacuation of the bladder, the cobra ureter may again be visible if the stenosis of the ostium is enough to dam back the renal secretion.

Associated with the ureterocœle there may be present some degree of hydronephrosis and hydroureter, and it is not unusual to find some congenital abnormality of the upper tract, such as bifid or double kidney, and double ureter. This last lends some colour to the congenital theory of causation of the condition.

Cases have recently been recorded, with full descriptions of the radiological features, by *Rohan Williams*, *Lenk* and *Akerlund*.

DIVERTICULUM OF THE URETER

This is an extremely rare condition. *Joseph* has described two cases, both arising from the lower end of the ureter, immediately above the interstitial portion. They are demonstrable by instrumental pyelography.

TUMOURS OF THE URETER

Carcinoma and papilloma are rare, and occur more often in the lower part



FIG. 382.—Left ureteroceles shown by intravenous urography. The lower ureter is dilated, and the wall of the ureteroceles is visible as a clear ring in the vesical shadow.

of the ureter. They may show a filling-defect in the ureterogram, but more commonly only a hydronephrosis or hydroureter is demonstrable.

TRANSPLANTATION OF THE URETERS

Grey Turner and *J. H. Saint* have published an account of the pyelographic appearances in six cases of transplantation of the ureters. The opera-

tions were performed for epispadias with incontinence in three cases, for ectopia vesicæ in two, and for post-partum vesico-vaginal fistula in one. Uroselectan B. urography was used in all.

The investigation showed that some degree of hydronephrosis and hydro-ureter is a common sequela of this operation. Only one ureter and four kidneys in the twelve were normal in appearance. Five of the ureters showed marked dilatation. *Grey Turner* and *Saint* consider that stenosis of the ureter at the point of transplantation is the major causative factor in the production of this dilatation, and that infection and kinking of the ureter where it enters the bowel may contribute to its production. Impaired renal function, shown by poor and delayed excretion of the drug, occurred *pari passu* with the dilatation. In no case, however, was the blood urea found to be higher than 48 mg. per 100 c.c. This, and the good general health of the patients reviewed, indicate that any ascending infection which occurred in them must have been mild in degree.

Storage of Urine.—The urography in these six cases showed that the rectum and colon both act as a urinary reservoir. In one case the drug-laden urine filled the colon as far back as the cæcum. six hours after the injection. This storage function explains how it is that these patients can retain urine for such long intervals.

CHAPTER XLI

THE BLADDER, PROSTATE, URETHRA, AND SEMINAL VESICLES

THE PRINCIPAL value of X-ray examination of the bladder is in the detection of vesical calculus. In some other cases, such as diverticulum or neoplasm, radiography may usefully supplement other means of diagnosis, but cystoscopy gives such accurate visual evidence of the condition of the bladder that where



FIG 383.—A normal Uroselectan B intravenous cystogram

it can be used, a cystogram becomes more of an academic demonstration than a diagnostic necessity. If for any reason cystoscopy is impossible or undesirable, cystography becomes of much greater importance, as the lesion which precludes the use of a cystoscope may allow the passage of a catheter, and if neither is permissible, intravenous, subcutaneous, or oral cystography is available.

The radiological methods available are plain and stereoscopic radiography in the antero-posterior, postero-anterior, and right and left oblique positions, radiography before and after emptying the bladder, and the various forms of cystography. The details of these have already been described, and their specific applications are indicated under the headings of the appropriate diseases.

NORMAL APPEARANCES

The outline of the upper two-thirds of the bladder, if distended, can be seen in almost every case, in a radiogram of satisfactory quality. This is of value in orientating vesical calculi, but gives no indication of other diseases of the bladder.

In the normal antero-posterior view the neck of the bladder lies just below the upper border of the symphysis pubis, and the fundus rises to a variable position above the symphysis, depending on the degree of distension; an average is $1\frac{1}{2}$ to 3 inches. The appearance in intravenous cystography has already been described (Fig. 383). Those of instrumental cystograms are similar, except that the density of the shadow is usually greater, and the bladder is usually distended to its globular form.

DIVERTICULA AND SACCULES OF THE BLADDER

Diverticula of the bladder are composed of pouches of vesical mucosa surrounded by fibrous tissue, fat, and occasionally some non-striped muscle.



FIG. 384 — Vesical diverticulum.

The communicating opening is small. They are classified as developmental and secondary.

DEVELOPMENTAL DIVERTICULA usually arise from the lateral or posterior wall of the bladder above the trigone, the opening being near a ureteral orifice. The ureter may actually open into the sac. They probably represent super-numerary ureteral buds.

SECONDARY DIVERTICULA (or saccules) are devoid of a musclic coat, are due to vesical obstruction, and are best termed saccules. They may be single or multiple, and vary in size from a pea to as large as or larger than the bladder itself (Fig. 384).

Calculi may develop in both types, with or without stones in the bladder—according to *Crenshaw and Crompton* in 12 per cent. of cases (Figs. 385–386).

Radiographic Features.—These are evident from the above account of the pathology of the condition. That cystography alone may not detect a small diverticulum requires emphasis. For the full investigation of this condition both cystoscopy and radiography are essential and complementary. Cystoscopy shows the orifice, and so the site, of a diverticulum, but can only hint at its size and contents. Cystography demonstrates its size, and plain radiography a stone in its lumen. An essential step in the examination is to take a further radiogram after emptying the bladder of its opaque contents, to demonstrate the presence or absence of stasis in the diverticulum.

Thus only cystoscopy combined with multi-plane cystography will give the full information necessary to determine the appropriate treatment.

TUMOURS OF THE BLADDER

Radiographic examination is of value when cystoscopy is impossible, as it sometimes is. If cystoscopy can be performed, the information given by it is so accurate compared with cystography as to make the latter of little account. The tumours commonly encountered are villous papilloma and carcinoma.

Villous Papillomata may be sessile or pedunculated, and vary in size from a pea to a tangerine orange. They are single in 60 per cent. of cases, and are usually situated towards the base of the bladder, just above the trigone. If large, they may be seen in a cystogram. The intravenous cystogram, by reason of its lesser density, is more likely to reveal a filling-defect caused by the tumour mass. Antero-posterior and oblique views should be taken, in order to obtain a profile view of the growth, if possible. The defect tends to have a wavy irregular margin, and the vesical capacity is not reduced, but evacuation may be incomplete if the tumour is near enough to the urethral orifice to act as a valve.

Carcinoma of the Bladder, apart from malignant degeneration of a villous papilloma, occurs in two radiographic types, nodular and infiltrating. In the nodular type the growth forms sessile excrescences, warty or smooth, projecting into the lumen of the bladder and giving rise to filling-defects in a cystogram (Figs. 387–388). These filling-defects tend to project less deeply into the lumen than do the papillomatous type, but often radiographic differentiation between the two is impossible.

The infiltrating type of carcinoma encroaches still less, but tends to contract the bladder wall.

In these tumours, as in the papillomata, intravenous cystography is more satisfactory than instrumental. By that method the involvement or otherwise of the ureter can often be demonstrated in a way impossible with the retrograde method.

A not uncommon occurrence is encrustation of a malignant vesical tumour.



FIG. 385.—Large calculus in the bladder and several small ones in vesical diverticulum.

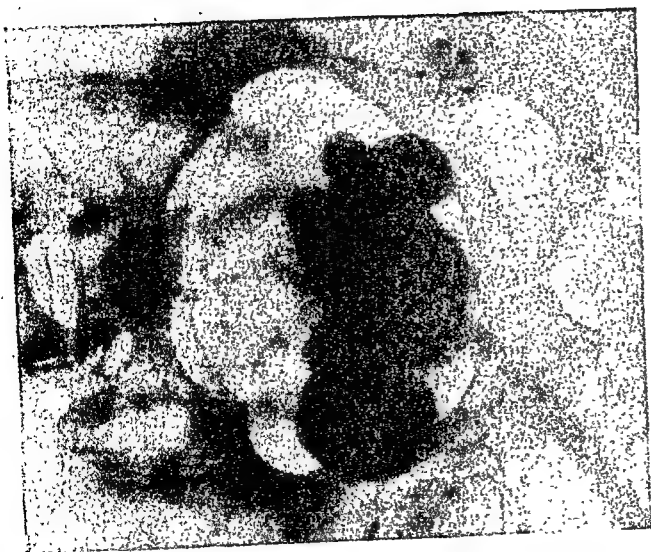


FIG. 386.—Cystogram of same case as Fig. 385.



FIG. 387.—Very extensive cauliflower carcinoma of the base of the bladder. Reflux of the contrast medium into the left ureter. (Instrumental cystogram.)



FIG. 388.—Urroselectan B cystogram of a carcinoma of the bladder, with incipient obstruction of the left ureter.

Such deposits are visible in a plain radiogram, and are significant inasmuch as they almost never occur in simple tumours.

The following table summarises the cystographic features of papilloma and carcinoma :

	<i>Papilloma</i>	<i>Carcinoma</i>
Vesical capacity	Normal	Diminished
Vesical outline	Normal	Distorted
Filling-defect	Irregular from presence of marginal villi	Irregular but dense and clear
Vesical evacuation	Often incomplete	Usually complete
Calcium encrustation	Never	Sometimes

Leiomyoma of the Bladder is very rare. It may vary in size from a tiny nodule to a large mass. When small, it is usually symptomless unless it involves the ureteric or urethral orifice. If large enough to project into or distort the bladder, it produces a smooth rounded filling defect in a cystogram, and cystoscopy shows the tumour to be covered with a mucosa.

HERNIATION OF THE BLADDER

Herniation of the bladder, e.g. into an inguinal hernia is capable of demonstration by cystography, which will show a deformed vesical contour communicating by a narrow channel with the herniated loculus. Cystocele is similarly demonstrable, though the necessity for such demonstration rarely, if ever, arises.

INFLAMMATION OF THE BLADDER

Cystitis.—A clue as to the presence of cystitis is given by the small vesical shadow in cases which result in contracture. In the rare cases in which encrustation of the mucosa occurs in cystitis, this is visible in a plain radiogram in the form of tenuous and diffuse mottling.

Tuberculous Cystitis.—It is said that tuberculosis of the bladder may be a primary lesion, but much more commonly it is secondary to tuberculosis of the kidney or genitalia.

The cystographic appearances depend on the macroscopic pathology of the infection. Tubercles form in the mucosa, caseate, and break down into tiny ulcers. These coalesce, and may form large deep craters. Irregular masses of granulation tissue occasionally develop.

As the process advances the bladder becomes contracted, trabeculated, and loculated (Fig. 389). In the earliest stages of the disease, therefore, cystography may show no abnormal signs. In the latter stages, the contracted irregular lobulated outline can be demonstrated by intravenous urography, a method which obviates the difficulties and risks of instrumental cystography.



FIG. 389.—Intravenous cystogram in a case of tuberculosis of the left kidney and bladder. The former fails to excrete the drug, and the latter is contracted and trabeculated

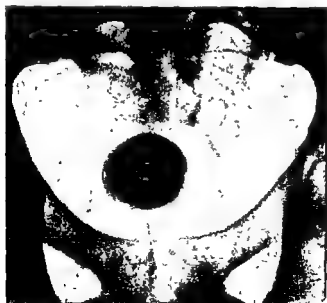


FIG. 390.—Laminated vesical calculus

VESICAL CALCULUS

Ætiology

These may be primarily or secondarily vesical.

True primary vesical calculi arise either in aseptic or infected urine, by deposition of salts on a nucleus, which may be a renal calculus, blood-clot, or foreign body. They are twenty times as common in men as in women. Infection and stagnation of the urine are important causative factors. Recurrent calculi in subjects over 50 usually indicate vesical obstruction, a condition which is present in 40 per cent. of all cases of vesical calculus.

Secondary vesical calculi are merely calculi which have passed down from the kidney. Usually voided in the urine (particularly in women), they nevertheless sometimes remain to form the nucleus of a primary vesical calculus. True secondary calculi form a very small proportion of the total—about 1 in 40.

Varieties.—The following types are met with:

URIC ACID CALCULUS.—

This is the commonest vesical calculus. It is composed of either pure uric acid, in which case it is invisible in a radiogram, or ammonium and sodium urates, when it casts a feeble shadow. These calculi are rounded or oval, smooth or finely nodular in contour, and frequently show lamination. They may reach a moderate size.

PHOSPHATIC CALCULUS.—This is the next commonest, and consists of calcium phosphate alone, or with ammonium-magnesium phosphate. They are soft, of rapid growth, moderately dense in shadow, and rarely show lamination. They form the largest type of stone.

CALCIUM OXALATE CALCULI form the third common type. They grow slowly, are very hard, do not reach a large size, and cast a very dense shadow, which may be "mulberry" or spiny in contour. The latter constitutes the "thorn-apple" calculus (Figs. 391–392).

RARE VESICAL CALCULI are calcium carbonate, cystin, xanthin, and

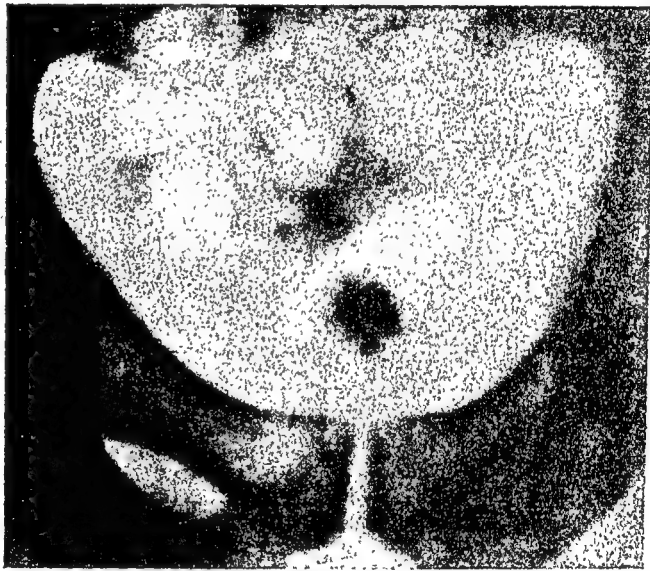


FIG. 391.—Calcium oxalate stone in the bladder, of the "thorn-apple" type.

indigo. Their degree of transparency has already been discussed under the section of renal calculi.

Vesical calculi are rarely pure.

Radiographic Features.—As in the case of vesical diverticula, a combination of cystoscopy and radiography may be necessary for the complete diagnosis of a stone in or near the bladder. In general, vesical calculi tend to lie in or near the midline, in the *bas fond*. If a number are present, some of them

may occupy a more lateral position, but if a single stone is seen to lie laterally, it may be in a diverticulum, a saccule, or the lower end of the ureter. This suspicion raised, it is a simple matter to take another radiogram after turning the patient on his other side and shaking him in the attempt to move the stone. This procedure is feasible only if the patient's bladder be full. The patient is then turned gently on to his back for the second radiogram. If the stone be free in the



FIG. 392.—Calcium oxalate vesical calculus, mulberry type

distended bladder, its position will have shifted. If it has not moved, the matter should be put to further proof by cystoscopy and urography.

In Great Britain vesical calculi rarely develop except in the presence of other urinary disease. The discovery of a stone in the bladder should therefore, as it is probably secondary to some other condition, lead the investigator to search for some further lesion in the kidneys, bladder, or prostate.

INVISIBLE CALCULI are more common in the bladder than in the kidney. They may, if large, be visible as negative shadows in a cystogram, but this is very uncertain compared with cystoscopy, an investigation which is usually decisive.

Differential Diagnosis.—A large number of other pelvic shadows have been enumerated. Many of them can be dismissed on naming them, others require serious consideration in arriving at a conclusion.

PHLEBOLITHS.—These have already been considered under the section on the ureter. The more centrally placed phleboliths may fall within the vesical area. Cystoscopy serves to exclude them.

FIG. 393.—Three vesical calculi and seven phleboliths.

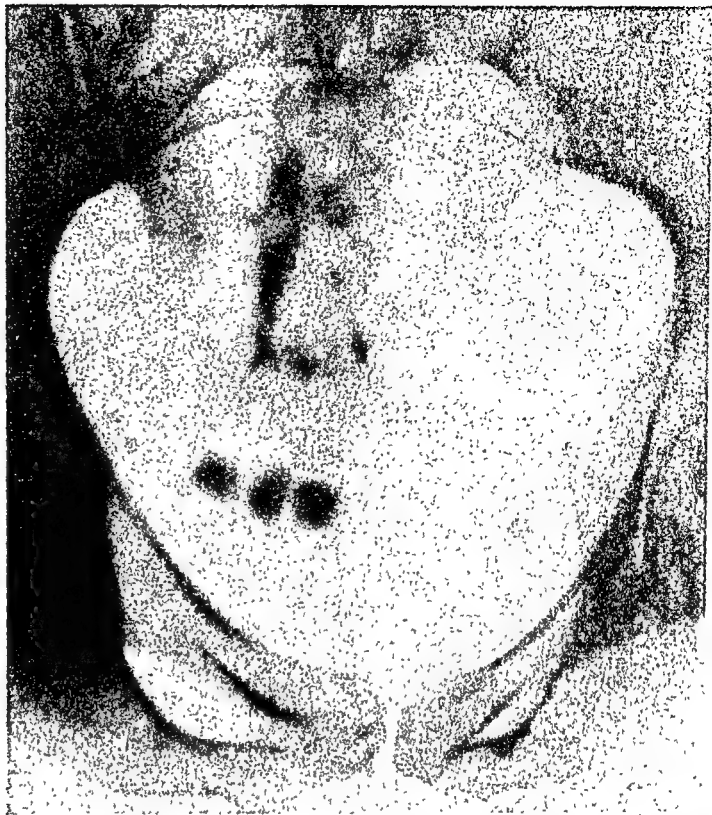
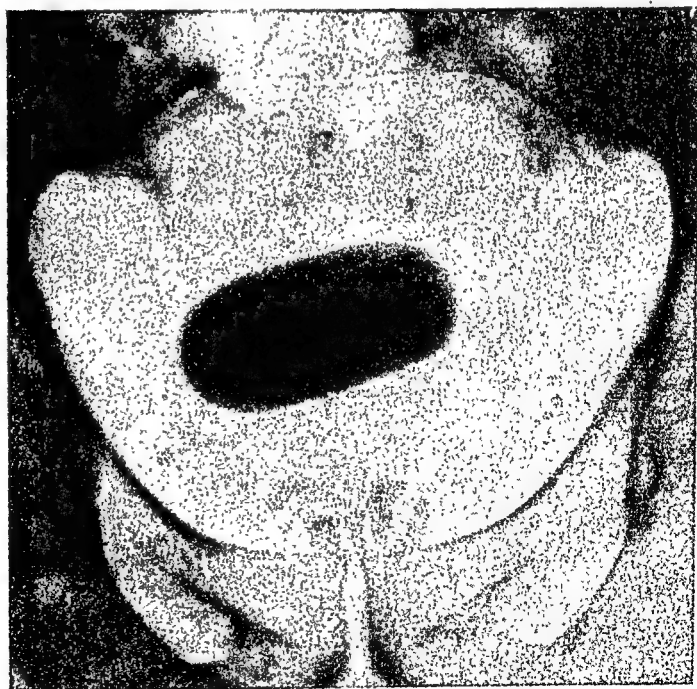


FIG. 394.—Large vesical calculus, and many primary prostatic calculi. Numerous phleboliths are present on each side of the pelvis.



PROSTATIC CALCULI.—The grouping of these and their position behind the shadow of the symphysis indicate their nature as a rule, failing which the prostatic clinical signs, and cystoscopy, if permissible, will give further differentiation.

URETERIC CALCULI.—Those situate in the lower end fall within the vesical limits, and may simulate a calculus in a diverticulum or pouch. Ureterography, intravenous or transvesical, and cystoscopy are the appropriate aids in diagnosis.

COPROLITHS AND RECTAL SCYBALA.—Their shadows are rather faint and irregular in texture, although they may present a rounded contour like a calculus. Re-examination after further preparation reveals their nature.

THE GLANS PENIS may cast a rounded, surprisingly dense shadow in the vesical area, and simulate a stone. This mistake should be avoided by recognition in the radiogram of the shadow of the corpus penis.

The following rarer shadows require but mention.

CALCIFIED FIBROMYOMA OF THE UTERUS.—This gives an irregularly striated shadow.

DERMOIDS AND TERATOMATA, containing bone or teeth

CALCIFICATIONS in the sacro-sciatic ligaments, iliac arteries, appendices epiploicæ, seminal vesicles, ano-coccygeal ligament, in an ectopic pregnancy, and in tuberculosis cystitis.

APPENDICULAR CONCRETIONS

VESICAL FISTULÆ

Of these, the supra-pubic and the vesico-vaginal do not require radiographic verification. In the vesico-enteric and vesico-colic fistulæ the portion of the bowel implicated may be shown by filling the bladder with sodium iodide solution. The passage of the opaque medium into the bowel may be observed fluoroscopically or in a radiogram. Care must be taken not to allow too much of the iodide solution to enter the bowel.

Those fistulæ due to difficult labour are commonly associated with damage to the kidney and ureter above. This arises from injury to the periureteric cellular tissue, with subsequent scarring and ureteric stenosis. The resulting hydronephrosis, hydrometer, and defective renal function can be well demonstrated by intravenous urography.

THE BLADDER IN NEUROLOGICAL LESIONS

The bladder has a double innervation, sympathetic and sacral autonomic. The former is inhibitory to micturition, and the latter motor. The rôle of the sympathetic, as shown by the results of lumbar sympathectomy is curious, in that if the operation is performed for bilateral hydronephrosis and hydro-ureter of sympathicotonic origin, a return to the normal may be expected. If, however, the operation is performed for vascular abnormalities of the lower

limbs, the vesical function being normal, no change in the latter results from the destruction of its sympathetic innervation.

The sympathetic fibres arise in the upper lumbar roots and reach the pelvic plexus via the aortic and hypogastric plexuses. The sacral parasympathetic fibres arise from the sacral roots and pass direct to the pelvic plexus.

Among the lesions of the central nervous system which may cause urinary disturbance are *tabes dorsalis*, *myelitis*, *spina bifida occulta*, and traumatic compression of the cord.

Tabes Dorsalis.—The bladder is affected at some stage of this disease in, it is said, 80 per cent. of cases.

The cystoscopic appearances in the tabetic bladder are often typical. Fine trabeculation of the bladder is common, with the exception of the trigone, which is, if anything, stretched and smooth. If the internal sphincter is paralysed, the trigone and prostatic urethra take a funnel shape. In a later stage the whole bladder may become dilated and atonic. The cystogram in the former stage may show the fundal trabeculation, and the funnel-shaped base, and in the atonic stage gross dilatation of the whole organ. The ureters and renal pelves may also show atonic dilatation, and reflux of the cystographic medium may fill the upper urinary tract.

Spina Bifida Occulta.—This is sometimes associated with disturbances of the vesical innervation. Clinically, urinary retention or incontinence may occur, and may not appear until adult life.

Two types of bladder may occur, according to the type of nerve defect: (1) A small trabeculated bladder, with a tendency to formation of diverticula, and (2) A dilated atonic bladder, with relaxed internal sphincter and funnel formation at the base. In both types there is a tendency to ureteral reflux, with development of hydronephrosis and hydroureter. The latter changes may be very gross.

Compression of the Cord and Myelitis.—The typical appearance in these conditions is atony of the bladder and ureters, relaxation of the internal sphincter, and ureteral reflux.

Atony of the Bladder.—In a certain number of cases of hypotonia or atony of the bladder, no evidence of a lesion of the central nervous system is present. This group, first described by *Thompson-Walker*, is supposed to be due to paralysis of the peripheral motor innervation. The vesical musculature is enfeebled, as tested by a manometer. The bladder is dilated, and the internal sphincter is relaxed.

It is evident that the cystographic appearances in the above nerve lesions are not differentially diagnostic, but tend to have features common to all. The points to be noted in the cystogram are:

- (1) The degree of vesical dilatation.
- (2) Trabeculation: this, when visible at all, causes a slight roughness or notching of the vesical contour.

(3) Paralysis of the internal sphincter, indicated by a funnel-shaped base of the bladder.

(4) Ureteric reflux, and the degree of hydronephrosis and hydroureter.

THE PROSTATE

In the usual radiogram of the vesical area the prostate lies behind the pubes. In order to throw its shadow clear of that of the pubic bones the central ray should be directed more in the axis of the pelvis—i.e. it should be given a caudal inclination of 10–15 degrees. The normal prostate cannot be seen in either a plain radiogram or a cystogram.

Enlarged Prostate.—Intravesical enlargement of the prostate is well shown in a cystogram in the form of a rounded indentation of the vesical shadow at its neck (Fig. 395).

After Prostatectomy the healing of the prostatic bed can be demonstrated by the progressive shrinkage of the initial irregular funnel shape as seen in a cysto-urethrogram (Fig. 396).

Carcinoma and Adenoma of the prostate may give signs in a urethrogram of their presence by a lengthening and distortion of the prostatic urethra, and by their protruding into the base of the vesical lumen. It is not possible to distinguish between them radiographically, unless there are osseous metastases present in the pelvic and other bones.

Prostatic Calculi

(1) **PRIMARY.**—These occur in the middle-aged and the elderly. According to *Forssell*, they have not been recorded in subjects younger than 47, but they are almost always present at autopsy in old men. In size they vary between a hemp seed and a pea. They are usually multiple and bilateral, and may be faceted. As many as 130 have been found in a prostate. From six to twelve is a common number to find. They arise in the corpora amylacea, and do not cause symptoms *per se*. When symptoms are present, they are due to an associated fibrous prostatitis.

(2) **SECONDARY.**—Rarely, prostatic calculi reach the size of a hazel nut. These are of urinary origin, develop in septic or alkaline urine, and are deposited in cavities left by prostatic abscesses. Because of the symptoms they produce, they are of considerable practical importance.

(3) **IMPACTED.**—A vesical or renal stone impacted in the prostatic urethra may be forced into the prostate.

Differential Diagnosis.—Prostatic calculi have to be distinguished in a radiogram from calcification in the prostate, in which the opacity tends to be striated in a fan shape from below upwards. It may form an almost complete cast of the prostate.

Phleboliths, calcified seminal vesicles, and calcification of the cartilage of the symphysis pubis are other conditions which might simulate prostatic calculi.



FIG. 395.—Cysto-urethrogram in a case of enlarged prostate.



FIG. 396.—Cysto-urethrogram of the prostate bed after prostatectomy : small saccules are seen on the posterior bladder wall.

THE URETHRA

Urethral stones, stones in fistulae, calculi under the prepuce, and foreign bodies are occasional objects to be seen in a plain urethral radiogram.

Urethrography forms a valuable means of investigation in certain urethral and prostatic conditions. It consists in radiography of the urethra after filling its lumen with an opaque medium. The technique of *Konstam* and *Cave* is suitable.

Lipiodol has been used as a contrast medium, but 12—15 per cent. sodium iodide is more convenient. It is injected through a urethral nozzle. The spongy or anterior urethra fills first, and when this is filled a sense of resistance can be felt—that of the anterior or external sphincter. If it is desired to examine the anterior urethra only, the end of the penis may be compressed with a clip and the radiograms taken. But if, as is usually the case, the posterior urethra also calls for investigation, the resistance of the external sphincter must be overcome by slight increase in the syringe pressure. When this sphincter has been overcome, the posterior urethra fills, but tends to empty itself into the bladder. It is essential, therefore, to keep up the pressure of the injection throughout the exposure, to replace the solution leaking into the bladder. If this is not done, the membranous and prostatic portions will not be visualised.

The radiograms should therefore be taken as quickly as possible. Postero-anterior views are unsatisfactory, as a portion of the urethra is seen end on. An oblique view, with the right buttock on the film and the tube centred just to the left of the symphysis pubis, shows the urethra throughout the whole extent of its S-curve. Alternatively, the patient may be turned 30 degrees on to his side, the under thigh fully flexed and abducted, the upper thigh hyperextended. The central ray is directed to the root of the penis, which lies along the under thigh.

Radiographic Appearance of the Normal Urethra.—

The spongy portion shows a uniform calibre of about $\frac{1}{4}$ to $\frac{1}{2}$ inch in its distal portion, gradually widening out into a bulbous dilatation just short of the membranous urethra. Here it abruptly narrows to join the membranous portion. The latter is short (about $\frac{1}{2}$ inch long) and narrows to a point as it reaches the deep triangular ligament to join the prostatic portion. Although the prostatic portion is very distensible, it retains a straight linear or slightly spindle-shaped appearance in a urethrogram, because of leakage into the bladder.

FIG. 397.—Diagram of a normal cysto-urethrogram

It is 1—1½ inches in length. It joins the base of the bladder at right angles, and does not normally show any funnel-shaped widening (Fig. 397).

Pathological Conditions.—The appearances in prostatic enlargement, prostatic tumours, and after prostatectomy have already been indicated.

A STRICTURE shows as a permanent filling-in defect in the urethral lumen. The upper limit can be shown in a radiogram taken while the patient is passing *per urethram* opaque fluid previously introduced into the bladder.

URETHRAL FISTULÆ can usually be seen clearly. If the sodium iodide leaks away too freely, lipiodol may be used and/or the sinus first occluded with adhesive plaster.

CALCULI AND FOREIGN BODIES are usually visible in the spongy portion in a plain radiogram. In the posterior urethra the opaque injection serves to show their position in that canal. A calculus may show in negative relief, and may be outlined by the medium adhering to it after the injection has been passed.

SEMINAL VESICLE, VAS DEFERENS AND EPIDIDYMIS

The seminal vesicles may be examined by plain radiography or by vesiculography. In a plain radiogram tuberculous calcification is visible, but is likely to be mistaken for a similar change in the prostate. The same applies to the rare cases of calculi in the vesicles.

The technique of vesiculography and vasography have already been described (p. 498), together with its indications. It is a method but rarely used. Methylene blue may be injected advantageously into the vas before the opaque material—its arrival in the urethra serves as a preliminary indication of patency (Fig. 398).

The patency of the tubes of the epididymis may be investigated by vasotomy followed by injection of lipiodol or other opaque medium.



FIG. 398.—Lipiodol vasogram in the normal subject.

REFERENCES

- ABESHOUSE, B. S., *Amer. J. Surg.*, 1934, XXV, 427.
ÅKERLUND, Å., *Acta. Radiol.*, 1935, XVI, 39.
BEER, E., and THEODORE, F. H., *J. Amer. med. Ass.*, 1934, CIII, 181.
BEGG, R. C., *Brit. J. Surg.*, 1936, XXIV, 18.
BIEBERBACH, W. D., COOK, P. H., and GOODALE, R. H., *Amer. J. Roentgenol*, 1934, XXXI, 778.

- BINZ and VON LICHTENBERG, A., *Radiology*, 1930, XV, 664.
- BOTEZ, G., *J. d'Urol.*, 1912, I, 193.
- BRAASCH, W. P., "Urography," Philadelphia, 1928.
- BRUNN, A. VON, *Arch. mikr. anat.*, 1893, XLI, 294.
- BUTZENFINGER, O., *Röntgenpraxis*, 1931, III, 881.
- CARELLI, H. H., and SORDELLI, E., *Rev. assoc. med. Argent.*, 1921, XXXIV, 421.
- CARLIER, V., and GERARD, M., *Rev. Chir. Paris*, 1912, XLVI, 9.
- CRENSHAW, J. L., and CROMPTON, C. B. R., *J. Urol.*, 1922, VIII, 185.
- FORSELL, G., *Munch. med. Wschr.*, 1909, LVI, 1176.
- GARDNER, R. A., and HEATHCOTE, R. St. A., *Brit. J. Radiol.*, 1932, V, 836.
- GUTIERREZ, R., *Amer. J. Surg.*, 1932, XV, 132.
- HARRIS, S. H., *Proc. R. Soc. Med.*, 1935, XXVIII, 1497.
- HELLMER, H. (quoted by GRAHAM HODGSON), *Trans. med. Soc. Lond.*, 1935, LVIII, 159.
- HIGHAM, A. R. C., *Brit. J. Surg.*, 1937, XXV, 139.
- HILDEBRAND, H., *Zbl. Chir.*, 1932, LIX, 1048.
- HINMAN, F., and LEE-BROWN, R. K., *J. Amer. med. Ass.*, 1924, LXXXII, 607.
- JFCK, H. S., *Trans. Sect. Urol. Amer. med. Assoc.*, 1931, 145.
- JONA, J. L., *Proc. R. Soc. Med.*, 1936, XXIX, 623.
- JOSEPH, E., and PERLMANN, S., "Die Harnorgane im Röntgenbild," Leipzig, 1931.
- KELLY, H. A., and BURMAN, C. F., "Diseases of Kidneys, Ureters and Bladder," New York, 1922.
- KLOSE, B., *Dtsch. z. Chir.*, 1904, LXII, 613.
- KÖHLER, A., "Röntgenology," London, 1935.
- KONSTAM, G. L. S., and CAVE, E. H. P., *Brit. J. Radiol.*, 1925, XXX, 121.
- LEGUEU, P., "La Pyeloscopie," Paris, 1927.
- LENK, E., *Fortsch. a. d. geb. Rönt.*, 1935, LI, 391.
- LICHTENBERG, A. VON, *Brit. J. Urol.*, 1931, III, 110.
- MACALPINE, J. B., "Cystoscopy and Urography," Bristol, 1935.
- MUSCHAT, M., *J. Urol.*, 1926, XVI, 351.
- NICAISE, V., *Enc. f. Urol.*, 1914, III, 75.
- QUINBY, W. C., *J. Urol. Balt.*, 1923, IX, 13.
- RAVASINI, C., *Urol. ital. Rev.*, 1934, XXXVIII, 851.
- ROBINSON, R. H. O. B., *Proc. R. Soc. Med.*, 1936, XXIX, 809.
- ROSENSTEIN, P., *Z. Urol.*, 1921, XV, 447.
- ROWNTREE, L. G., and OTHERS, *J. Amer. med. Ass.*, 1923, LXXX, 368.
- SCHUMACHER, P., *Arch. Gynaek.*, 1930, CXLI, 28.
- SCHWICK, M., *J. Amer. med. Ass.*, 1933, CI, 1853.
- SCHWICK, M., and JACHES, L., *Radiology*, 1934, XXIII, 216.
- THOMPSON, A. R., *Guy's Hosp. Rep.*, 1929, LXXIX, 201.
- THOMSON-WALKER, J. W., Choyce's "System of Surgery," London, 1932, II.
- TURNER, G. GREY, and SAINT, J. H., *Brit. J. Surg.*, 1936, XXIII, 580.
- WILLIAMS, E. ROHAN, *Brit. J. Radiol.*, 1936, IX, 59.

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